DISSERTATION ON

STUDY OF LIVER ABSCESS AND VARIOUS MODALITIES OF MANAGEMENT OF AMOEBIC LIVER ABSCESS

M.S.DEGREE EXAMINATION
BRANCH – I
GENERAL SURGERY

THANJAVUR MEDICAL COLLEGE AND HOSPITAL

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI

MARCH – 2009

CERTIFICATE

This is to certify that dissertation entitled '**Study of liver abscess and various modalities of management of amoebic liver abscess'** is a bonafide record of work done by **Dr.DHANDAYUTHAPANI.V,** in the Department of General Surgery, Thanjavur Medical College, Thanjavur, during his Post Graduate Course from 2006-2009 under the guidance and supervision of **PROF. DR.S.MOHAMMED ISMAIL M.S.,** and **PROF. DR.G. AMBUJAM, M.S. FICS.** This is submitted in partial fulfillment for the award of **M.S. DEGREE EXAMINATION-BRANCH I (GENERAL SURGERY)** to be held in March 2009 under the **Tamilnadu Dr. M.G.R. Medical University, Chennai.**

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DECLARATION

I declare that this dissertation entitled '**A Study of liver abscess and various modalities of management of amoebic liver abscess'** is a record of work done by me in the department of General Surgery, Thanjavur medical college, Thanjavur, during my Post Graduate Course from 2006-2009 under the guidance and supervision of my unit Chief PROF**. DR. S.MOHAMMED ISMAIL,M.S.,** and professor and head of the department **PROF. DR. G. AMBUJAM, M.S., FICS.** It is submitted in partial fulfillment for the award of **M.S. DEGREE EXAMINATION- BRANCH I (GENERAL SURGERY)** to be held in March 2009 under the **Tamilnadu Dr. M.G.R. Medical University, Chennai.** This record of work has not been submitted previously by me for the award of any degree or diploma from any other university.

Dr.DHANDAYUTHAPANI.V.

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STUDY OF LIVER ABSCESS AND VARIOUS, MODALITIES OF MANAGEMENT OF AMOEBIC LIVER ABSCESS.

CONTENTS

INTRODUCTION

Liver abscess is an entity that continues to pose diagnostic and therapeutic problem. Two major types of Liver abscess are amoebic those caused by Entamoeba histolytica and pyogenic those caused by bacteria. Others include fungal and TB abscess that are observed more commonly in Immuno compromised patients.

In many countries amoebiasis and amoebic dysentery continue to be common health problems. Amoebic infection of colon, liver and other organs are common in area of poor sanitation and particularly in developing countries with contaminated water supply and poor hygiene.

Amoebic liver abscess aptly called as tropical liver abscess as it’s more common in tropics. Typically these endemic areas of amoebiasis are located in tropical and subtropical areas of world. The early description of amoebiasis came from India only.

Careful history and skillful clinical evaluation may provide important information about the diagnosis. (1) USG Abdomen and (2) Serological test are the most important other methods of evaluation.

INVESTIGATIONS IN THE DIAGNOSIS OF LIVER ABSCESS:

The management of amoebic liver abscess involved with recognition of colonic amoebiasis as the antecedent source of the liver infection. Early treatment by open surgical drainage alone had met with only limited success. Efforts to treat both liver abscess and colonic infestation improved the response rates. Eventually the development of systematic amoebicidal agents compiled with closed aspiration became the treatment of choice for amoebic abscess with majority of patients cured.

Though the amoebic liver abscess is treated conservatively with the drugs alone there are still few instances in which radiological and surgical interventions are required. This study is mainly focused on the comparison of various modalities of treatment of amoebic liver abscess and to find the most appropriate and cost effective treatment in the management of amoebic liver abscess.

SURGICAL ANATOMY

EMBRYOLOGY:

The liver primordium appears in the middle of the 3rd week of gestation as on outgrowth of endodermal epithelium at the distal end of the foregut. This bud grows into the ventral mesogastrium and passes through it into the septum transversum. It enlarge soon shows a division into layers. Cranial part called as the pars hepatica and smaller caudal portion called as the pars cystica. The pars hepatica divides into right and left part of each of which later develops in to right and left lobe of liver.

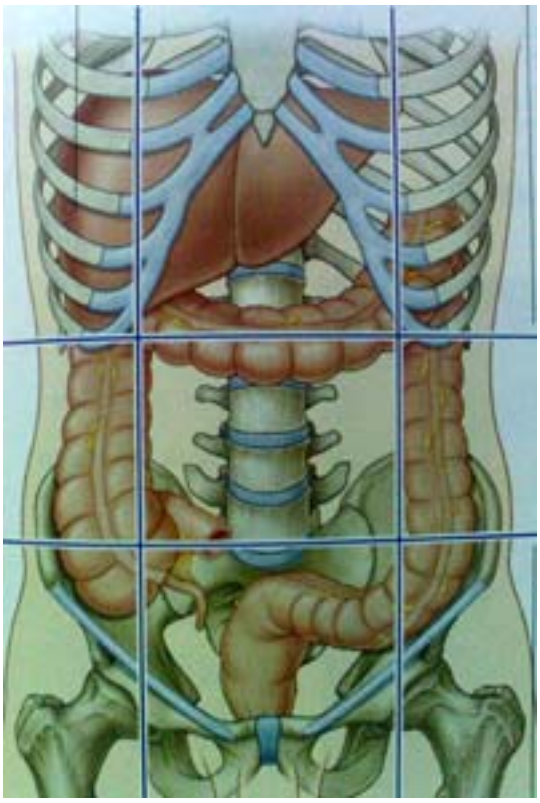
The cells arising from this division are broken up into the interlacing columnar cells called hepatic trabeculae. In this process the umbilical vein and vitelline vein are broken up to form the sinusoids of liver. Sinusoids are also formed from the mesenchyme of the septum transversum.

The endodermal cells of the hepatic bud gives rise to the parenchyma of the liver and to bile capillaries. The mesoderm of the septum transversum forms the capsules and fibrous tissue of base of liver.

ANATOMY OF LIVER:

The liver is the largest gland in the body and, after the skin, the largest single organ. It weighs approximately 1500 g and accounts for approximately

SURFACE ANATOMY OF LIVER



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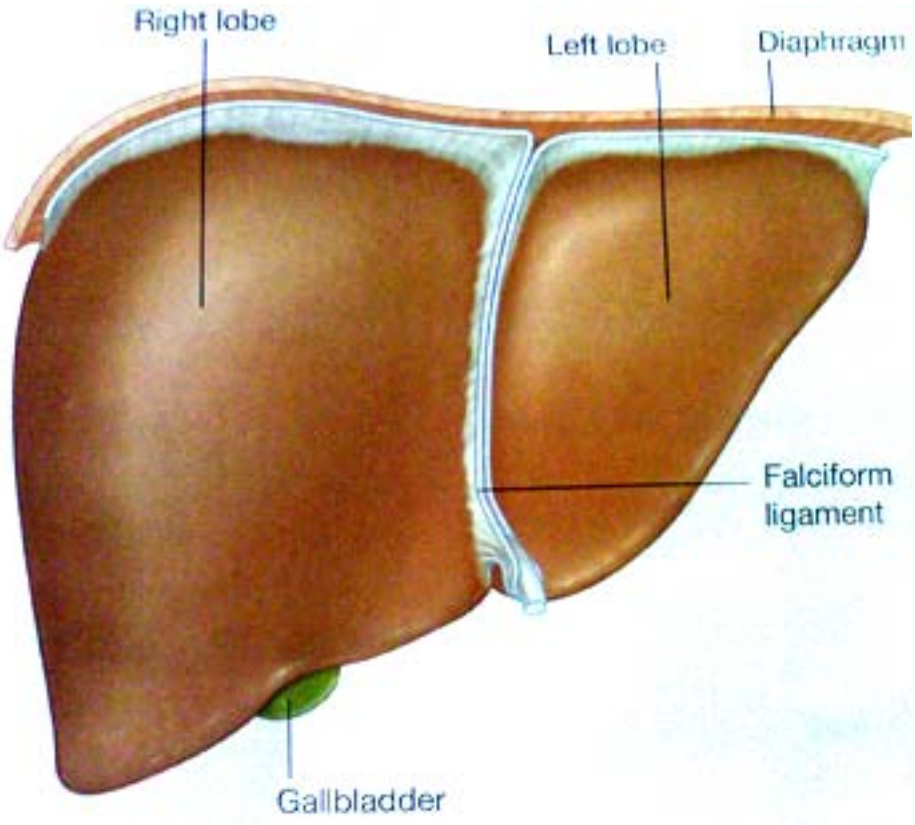

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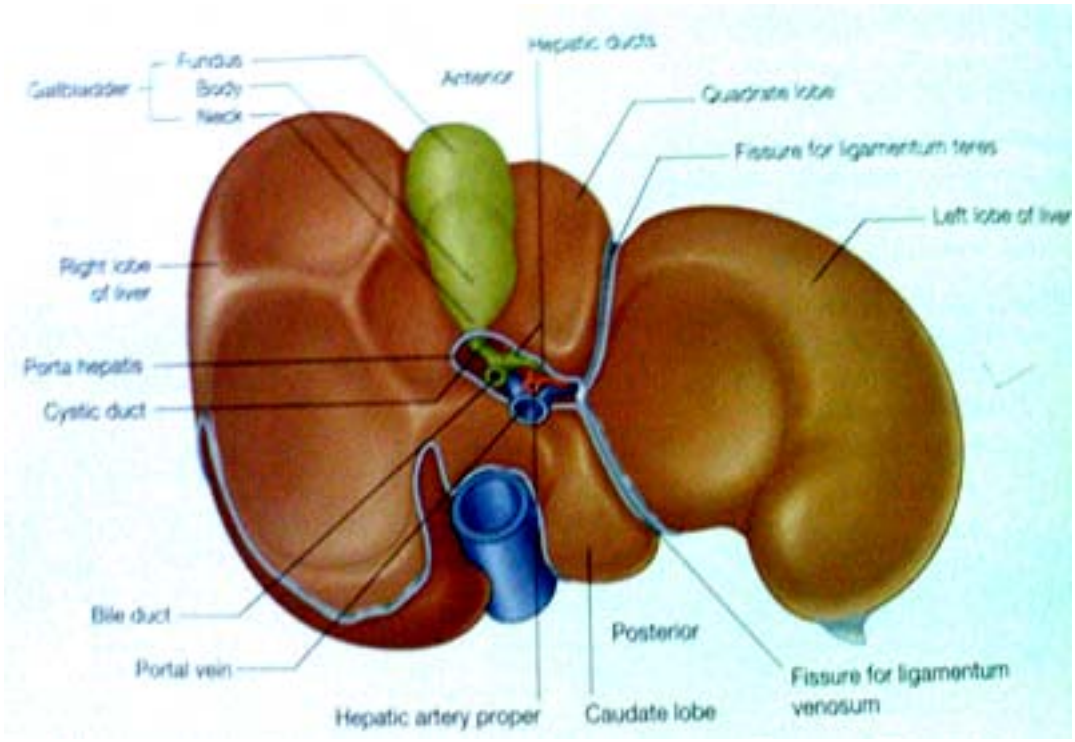
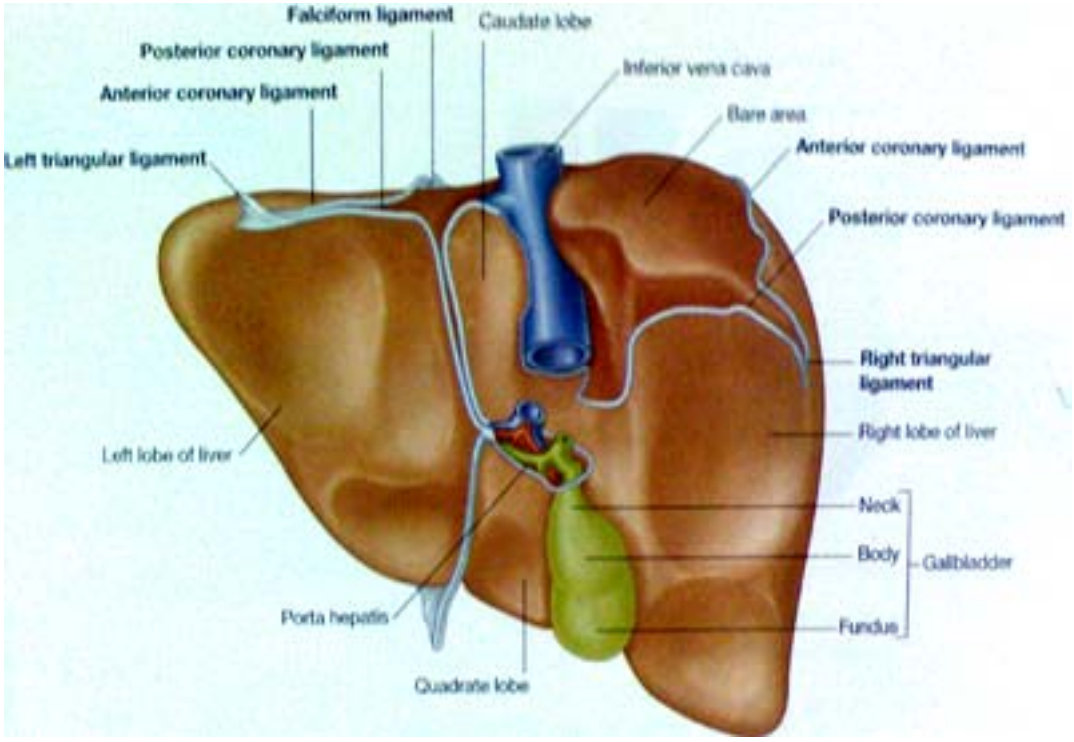


ANATOMY OF LIVER



2.5% of adult body weight. In the late fetus it also serves as a hematopoietic organ. It is proportionately twice as large (5% of body weight). From early childhood onwards, it occupies almost all of the right hypochondrium and epigastrium. It extends into the left hypochondrium, inferior to the diaphragm, which separates it from the pleura, lungs, pericardium, and heart.

Except for fat, all nutrients absorbed from the gastrointestinal tract are initially carried to the liver by the portal venous system. In addition to its many metabolic activities, the liver stores glycogen and secretes bile. Bile passes from the liver via the biliary duct. Right and left hepatic ducts join to form the common hepatic duct, which unites with the cystic duct to form the common bile duct. The liver produces bile continuously; however, between meals it accumulates and is stored in the gallbladder, which also concentrates the bile by absorbing water and salts. When food arrives in the duodenum, the gallbladder sends concentrated bile through the bile ducts to the duodenum.

Surfaces, Peritoneal Reflections, and Relationships of the Liver:-

The liver has a convex diaphragmatic surface (anterior, superior, and some posterior) and a relatively flat or even concave visceral surface

(posteroinferior), which are separated anteriorly by its sharp inferior border. The diaphragmatic surface of the liver is smooth and dome shaped, where it is related to the concavity of the inferior surface of the diaphragm.

Subphrenic recesses are superior extensions of the peritoneal cavity (greater sac) and exist between diaphragm and the anterior and superior aspects of the diaphragmatic surface of the liver. The subphrenic recesses are separated into right and left recesses by the falciform ligament, which extends between the liver and the anterior abdominal wall. The portion of the supracolic compartment of the peritoneal cavity immediately inferior to the liver is the subhepatic space. The hepatorenal recess (hepatorenal pouch; Morison pouch) is the posterosuperior extension of the subhepatic space, lying between the right part of the visceral surface of the liver and the right kidney and suprarenal gland. The hepatorenal recess is a gravity-dependent part of the peritoneal cavity in the supine position; fluid draining from the omental bursa flows into this recess. The hepatorenal recess communicates anteriorly with the right subphrenic recess "Recall that normally all recesses of the peritoneal cavity is potential spaces only, containing only enough peritoneal fluid to lubricate the adjacent peritoneal membranes.

Surface and peritoneal relationship of Liver:

A. This sagittal section through the diaphragm, liver, and right kidney demonstrates the two surfaces of the liver and related peritoneal recesses. B. The peritoneal reflections (ligaments) and cavity related to the liver are shown. The attachments of the liver are cut through and the liver is turned to the right and posteriorly, C. The anterior part of the domed diaphragmatic surface of the liver conforms to the inferior surface of the diaphragm. This extensive surface of the liver is divided into superior, anterior (shown here), right, and posterior parts. D. In the anatomical position, the visceral surface of the liver is directed inferiorly, posteriorly, and to the left. In embalmed specimens, impressions remain where this surface is contacted by adjacent structures. E. In the supine position, the hepatorenal recess is gravity dependent, receiving drainage from the omental bursa and upper abdominal (supracolic) portions of the greater sac.

The diaphragmatic surface of the liver is covered with visceral peritoneum, except posteriorly in the bare area of the liver where it lies in direct contact with the diaphragm. The bare area is demarcated by the reflection of peritoneum from the diaphragm to it as the anterior (upper) and posterior (lower) layers of the coronary ligament. These layers meet on the right to form

the right triangular ligament and diverge toward the left to enclose the triangular bare area. The anterior layer of the coronary ligament is continuous on the left with the right layer of the falciform ligament, and the posterior layer is continuous with the right layer of the lesser omentum. Near the apex (the left extremity) of the wedge-shaped liver, the anterior and posterior layers of the left part of the coronary ligament meet to form the left triangular ligament. The inferior vena cava traverses a deep groove within the bare area of the liver.

The visceral surface of the liver is covered with peritoneum except at the fossa for the gallbladder and the porta hepatica transverse fissure where the vessels (portal vein, hepatic artery, and lymphatic vessels), the hepatic nerve plexus, and hepatic ducts that supply and drain the liver center and leave it. In contrast to the smooth diaphragmatic surface, the visceral surface bears multiple fissures and impressions from contact with other organs. Two sagittal fissures, linked centrally by the transverse porta hepatis, form the letter H on the visceral surface. The right sagittal fissure is the continuous groove formed anteriorly by the fossa for the gallbladder and posteriorly by the groove for the vena cava; the left sagittal fissure is the continuous groove formed anteriorly by the fissure for the ligamentum teres or round ligament, and posteriorly by the fissure for the ligamentum venosum. The round ligament of the liver is the

fibrous remnant of the umbilical vein, which carried well-oxygenated and nutrient-rich blood from the placenta to the fetus; the round ligament and small paraumbilical veins course in the free edge of the falciform ligament. The ligamentum venosum is the fibrous remnant of the fetal ductus venosus, which shunted blood from the umbilical vein to the IVC, short-circuiting the liver (Moore and Persaud, 2003).

The lesser omentum, enclosing the portal triad (bile duct, hepatic artery, and portal vein) passes from the liver to the lesser curvature of the stomach and the first 2 cm of the superior part of the duodenum. The thick, free edge of the lesser omentum extends between the porta hepatis and the duodenum (the hepatoduodenal ligament) and encloses the structures that pass through the porta hepatis. The sheet-like remainder of the lesser omentum, the hepatogastric ligament, extends between the groove for the ligamentum venosum of the liver and the lesser curvature of the stomach.

Visceral surface of the liver:

A. The four anatomical lobes of the liver are defined by external features (peritoneal reflections and fissures). The left sagittal fissures (and the falciform ligament on the diaphragmatic surface) demarcate right and left

lobes. The right and left sagittal fissures and the porta hepatis connecting them form an H on the visceral surface, demarcating the quadrate and caudate lobes.B. Structures forming the fissures of the visceral surface are shown. The round ligament of the liver is the occluded remains of the fetal umbilical vein. The ligamentum venosum is the fibrous remnant of the ductus venosum that shunted blood from the umbilical vein to the IVC.

The anterior sagittal cut is made in the plane of the fossa for the gallbladder, and the posterior sagittal cut is in the plane of the fissure for the ligamentum venosum. These cuts have been joined by a narrow coronal cut in the plane of the porta hepatis. The portal triad passes between the layers of the hepatoduodenal ligament to enter the liver at the porta hepatis. The common hepatic artery passes between the layers of the hepatogastric ligament.

In addition to the fissures, impressions on the visceral surface reflects the liver's relationship to the:

- Right side of the anterior aspect of the stomach (the gastric and pyloric areas).
- Superior part of the duodenum (the duodenal area).
- Lesser omentum (extends into the fissure for the ligamentum

venosum).

- Gallbladder (fossa for gallbladder).
- Right colic flexure and right transverse colon (the colic area).
- Right kidney and suprarenal gland (the renal and suprarenal areas).

Subphrenic Abscesses

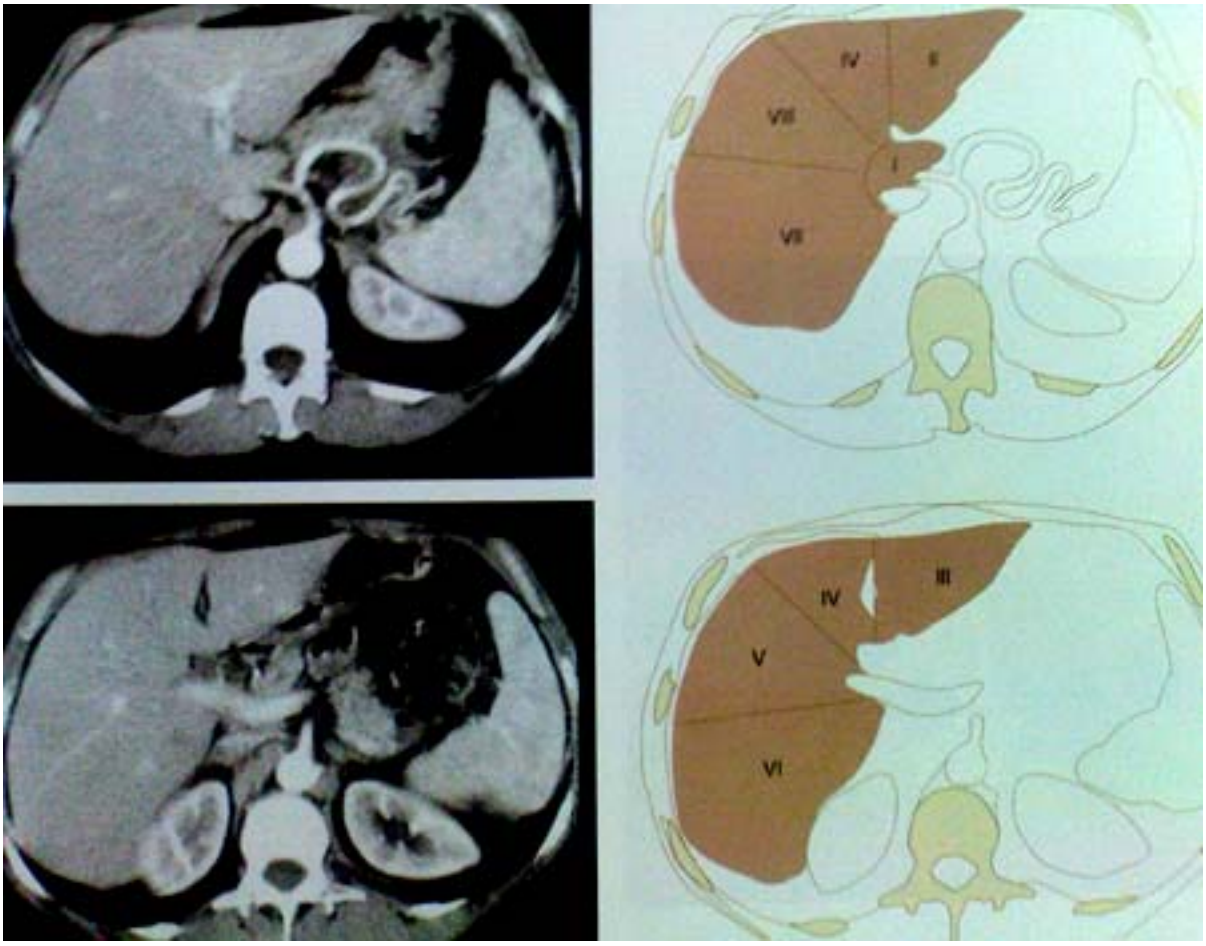
Peritonitis may result in the formation of localized abscesses in various parts of the peritoneal cavity. A common site for pus to collect is in a subphrenic recess or space. These subphrenic abscesses are more common on the right side because of the frequency of ruptured appendices and perforated duodenal ulcers. Because the right and left subphrenic recesses are continuous with the hepatorenal recess (the lowest [most gravity dependent] parts of the peritoneal cavity when supine), pus from a subphrenic abscess may drain into one of the hepatorenal recesses, especially when patients are bedridden. A subphrenic abscess is often drained by an incision inferior to, or through, the bed of the 12th rib (Ellis, 1992), obviating the formation of an opening in the pleura or peritoneum. An anterior subphrenic abscess is often drained through a subcostal incision located inferior and parallel to the right costal margin.

Anatomical Lobes of the Liver:

Externally, the liver is divided into two topographical (anatomical) lobes and two accessory lobes by the reflections of peritoneum from its surface, the fissures formed in relation to those reflections, and the vessels serving the liver and gallbladder. These superficial accessory lobes I are not true lobes as the term is generally used in relation to glands and are only secondarily related to the liver's internal architecture. The essentially midline plane defined by the attachment of the falciform ligament and the left sagittal fissure separates a large right lobe from a much smaller left lobe. On the slanted visceral surface, the right and left sagittal fissures surround and the transverse porta hepatis demarcates two accessory lobes (parts of the anatomic right lobe): the quadrate lobe anteriorly and inferiorly and the caudate lobe posteriorly and superiorly. The caudate lobe is so-named not because it is caudal in position but because it often gives rise to form of an elongated papillary process. A caudate process extends to the right, between the IVC and the portal hepatis, connecting the caudate and right lobes.

SEGMENTS OF LIVER WITH CT

CORELATION



Functional Subdivision of the Liver

Although not distinctly demarcated internally, where the parenchyma appears continuous, the liver has functionally independent right and left livers (parts or portal lobes) that are much more equal in size than the anatomical lobes; however, the right liver is still somewhat larger. Each part receives its own primary branch of the hepatic artery and portal vein and is drained by its own hepatic duct. The caudate lobe may in fact be considered a third liver; its vascularisation is independent of the bifurcation of the portal triad (it receives vessels from both bundles) and is drained by one or two small hepatic veins, which enter directly into the IVC distal to the main hepatic veins. The liver can be further subdivided into four divisions and then into eight surgically resectable hepatic segments, each served independently by a secondary or tertiary branch of the portal triad.

Blood Vessels of the Liver:

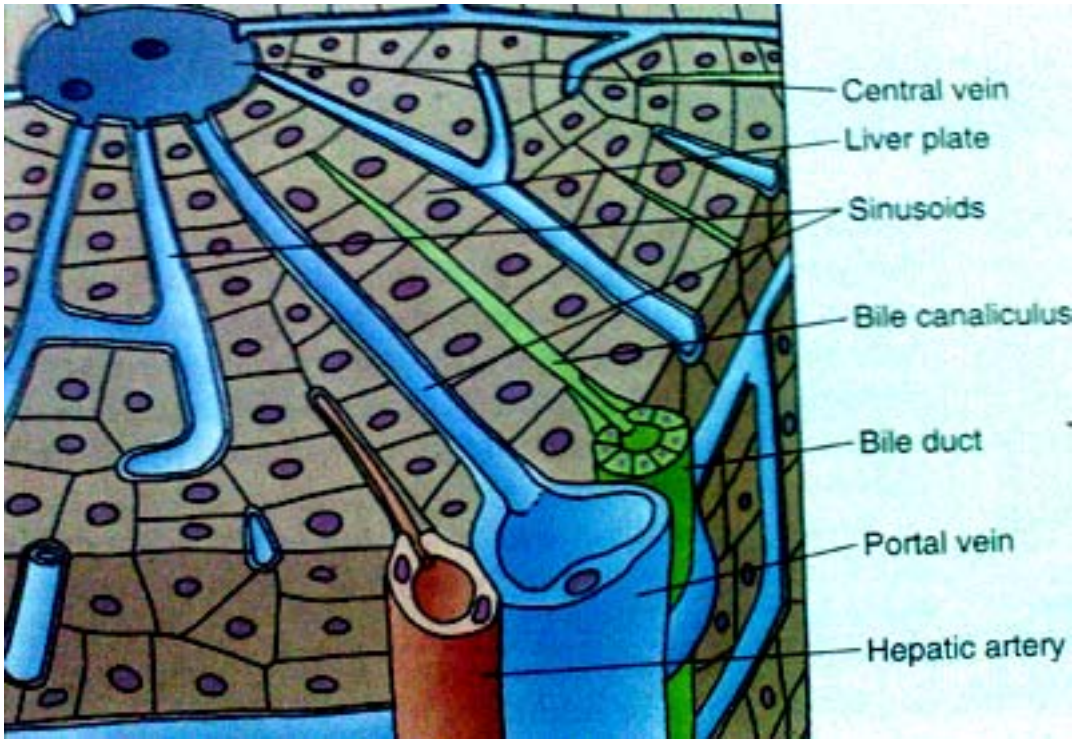
The liver, like the lungs, has a dual blood supply (afferent vessels): a dominant, venous source and a lesser arterial one. The portal vein brings 75-

80% of the blood to the liver. Portal blood, containing about 40% more oxygen than blood returning to the heart from the systemic circuit, sustains the liver parenchyma (liver cells or hepatocytes). The portal vein carries virtually all of the nutrients absorbed by the alimentary tract (except lipids, which bypass the liver in the lymphatic system) to the sinusoids of the liver. Arterial blood from the hepatic artery,

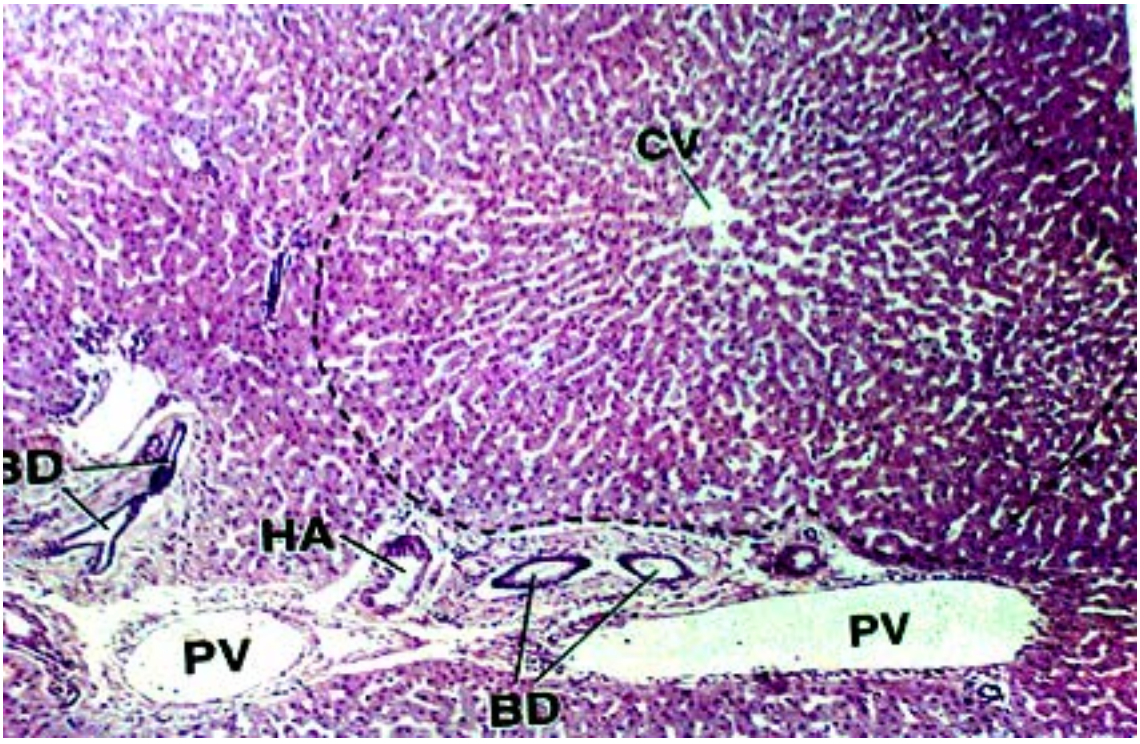
accounting for only 20-25% of blood received by the liver, is distributed initially to non-parenchymal structures, particularly the intrahepatic bile ducts. The portal vein, a short, wide vein, is formed by the superior mesenteric and splenic veins posterior to the neck of the pancreas and ascends anterior to the IVC as part of the portal triad in the hepatoduodenal ligament. The hepatic artery, a branch of the celiac trunk, may be divided into the common hepatic artery, from the celiac trunk to the origin of the gastroduodenal artery, and the hepatic artery proper, from the origin of the gastroduodenal artery to the bifurcation of the hepatic artery. At or close to the porta hepatis, the hepatic artery and portal vein terminate by dividing into right and left branches; these primary branches supply the right and left livers, respectively. Within each part, the simultaneous secondary branching of the portal vein and hepatic artery (portal pedicles) are consistent enough to supply the medial and lateral

divisions of the right and left liver, with three of the four secondary branches undergoing further (tertiary) branching to supply independently seven of the eight hepatic segments. Between the divisions are the right, intermediate (middle), and left hepatic veins, which are inter segmental in their distribution and function, draining parts of adjacent segments. The hepatic veins, formed by the union of collecting veins that in turn drain the central veins of the hepatic parenchyma, open into the IVC just inferior to the diaphragm. The attachment of these veins to the IVC helps hold the liver in position.

ANATOMY OF LIVER



HISTOLOGY OF LIVER



HISTOLOGY:

The substance of liver is made of liver cells arranged in the form of hexagonal area called as hepatic lobules. The lobules are partially separated by connective tissue. Each lobule has a central vein, from which numerous sinusoids passes radially. The space between the sinusoids is occupied by liver cells. The plates of liver cells look like cords when seen in section. Along the periphery of lobules there are angular intervals filled by connective tissue. Each such area contains a branch of portal vein, a branch of hepatic artery, and an inter lobular bile duct. These three constitutes portal triad.

REVIEW OF LITERATURE

AMOEBIC LIVER ABSCESS:

Incidence

It has highest incidence in tropical and subtropical climates and in areas with poor sanitation.

Hepatic liver abscess occurs in only 3.77 of all patients with intestinal amoebiasis.

Mean age of the patient is 3rd and 4th decade. There is no racial predisposition. Male to female ratio is 9:1

History

In 1875, LOSCH discovered that Entamoeba histolytica was the causative factor of amoebic dysentery.

In 1890 SIROSLER first reported the presence of amoeba in liver abscess as well as in the stools of some patients. In 1891 COUNCILMAN and LAFLER first used the term “AMOEBIC LIVER ABSCESS”}

In 1992, ROGERS demonstrated that active amoeba were present infrequently in the pus but were found in the wall of amoebic liver abscess.

ETIOLOGY AND PATHOGENESIS:

The Cystic form of E- histolytica is the infecting agent, usually through contaminated water orally. They are resistant to acid PH and drying, digested by trypsin in small intestine and few invasion trophozoites are released which multiply in caecum.

Trophozoites exist in two forms namely small and Large, where tissue invasion occurs trophozoites digest erythrocyte and become large forms. The most frequent site of extra intestinal colonization is liver. The three possible routes are (1) via portal vein (2) via lymphatics (3) by direct extension, but mostly through portal vein.

In liver, trophozoites lodge in small vessels and produce thrombosis and of small areas of hepatic parenchyma. At first the necrotic material consists of a solid slough and later the centre liquefies by cytolytic action of amoeba and liquefaction extends radially.

The syndrome of slightly enlarged and tender liver, right upper quadrant pain, intermittent fever and Leukocytosis in patients with

amoebic dysentery is something referred to as diffuse amoebic hepatitis. Healing at this stage or of further a progression is decided by host's immune status and hosts nutrition. If it progresses coalition of number of small areas of necrosis produce macro abscess.

The pus is dark reddish brown in colour (anchovy sauce) sterile consisting of mixture of blood and destroyed tissues. Trophozites are rarely found in the pus but in the wall of the cavity.

Abscess cavity varies in size from 1 to 25cm. Leucocytes infiltration and inflammatory reactions are characteristically absent. If untreated will rupture into adjacent organs like peritoneum, pleural cavity and pericardium.

It is more likely to be solitary and located in the right lobe poster superior aspect of liver.

HOST FACTORS:

The human host represents the major reservoir. Interpersonal transmission occurs via flies and handlers and by sewage contamination of water sources. Male homosexuals also transmit the disease.

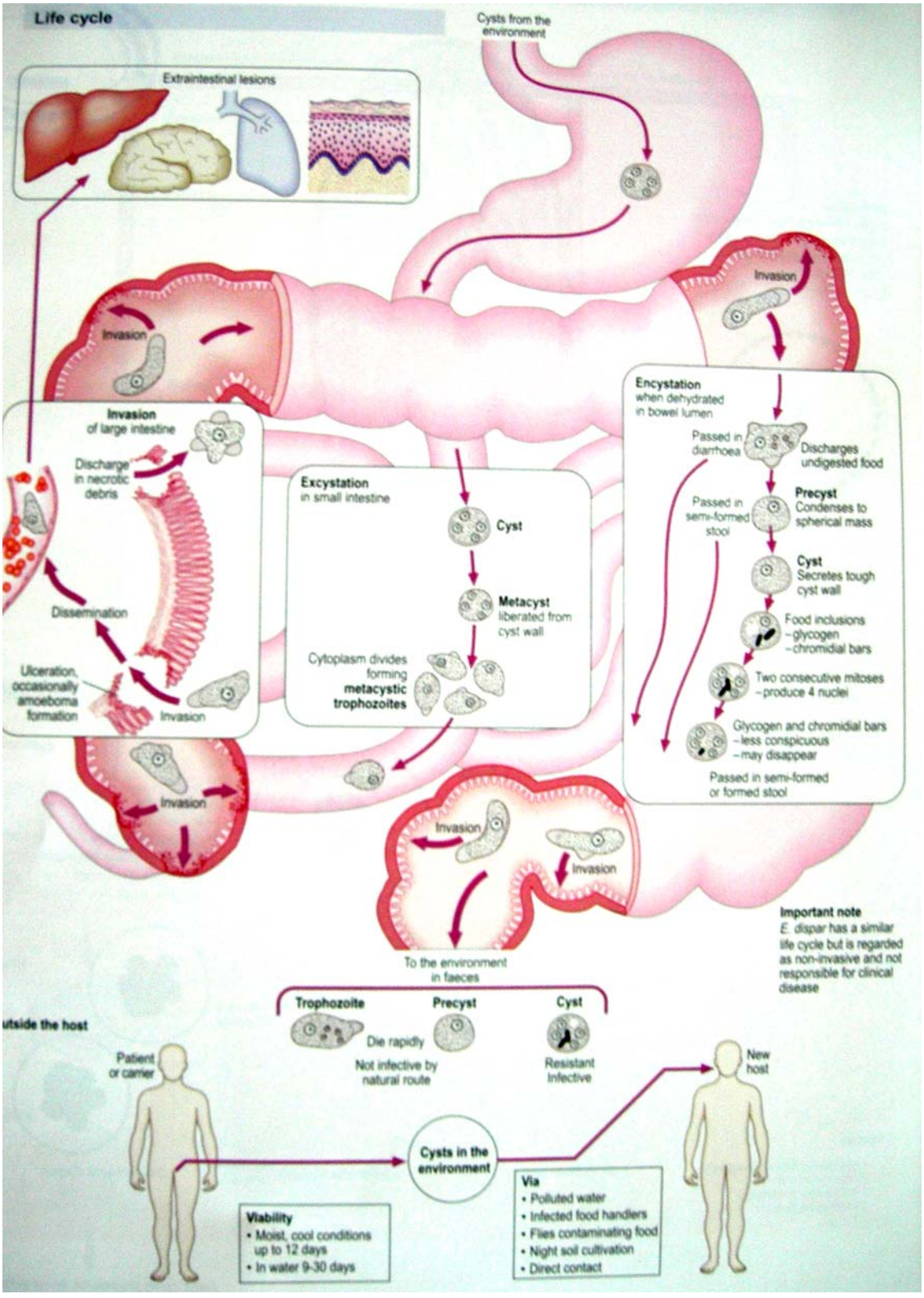
Menstruating women, breast fed children have low incidence of invasive infection due to presence of protective IgA, in the immune mother's milk, and due to the low iron content of milk. High content of Iron in the diet, high carbohydrate with decreased immunity predisposes to invasive Amoebiasis.

LIFECYCLE OF ENTAMOEBA HISTOLYTICA:

E.histolylica passes its life cycle only in one host, the man. There are mainly two phases of development, trophozoites and cyst with a transitory stage of pre-cystic form. The mature quadrinucleate cysts are the infective forms of the parasite. When these cysts are swallowed along with the contaminated food and drink by a susceptible person, they are capable of further development inside his gut. The fully developed cysts, thus gaining entrance into the alimentary canal, pass unaltered through the stomach. The cyst-wall is resistant to the action of the gastric juice but is digested by the action of trypsin in the intestine. The excystation occurs when the cyst reaches the caecum or the lower part of the ileum (neutral or slightly alkaline medium). During the process, the cytoplasmic body retracts and loosens itself from the cyst-wall. Vigorous amoeboid

movements cause a rent to appear in the cyst-wall through which at first a small mass of cytoplasm and then ultimately the whole body comes out. Each cyst liberates a single amoeba with four nuclei, a tetranucleate amoeba which eventually forms eight amoebulae {metacystic trophozoites) by the division of nuclei with successive fission of cytoplasm. The young amoebulae being actively motile, invade the tissues and ultimately lodge in the sub mucous tissue of the large gut, their normal habitat. Here they grow and multiply-by binary fission. It is to be noted that the trophozoites phase of the parasite is responsible for producing the characteristic lesion of Amoebiasis.

LIFE CYCLE OF ENTAMOEBA HISTOLYTICA



During growth, E. histolytica secretes a proteolytic ferment of the nature of histolysin which brings about destruction and necrosis of tissues and thereby helps the parasite in obtaining nourishment through absorption of these dissolved tissue juices. The tissue-invading amoebae gradually recede from the dead tissues towards the margin of healthy ones and in this way the trophozoites of E. histolytica often wander about in the tissues of the gut-wall, entering into deeper layers and may sometimes actually find their way into the radicles of the portal vein to be carried away to the liver where their further progress may be arrested. In the liver the trophic forms may for a time grow and multiply but encystation does not occur. Hence such an invasion is always to be looked upon as an accident on the part of the parasite because so far as its biological aspect is concerned it has reached a dead end.

Those parasites that remain in the intestinal wall may cause an attack of acute dysentery (ulcerative colitis) in which a large number of trophozoites are discharged along with the slough. This again is a loss to the species itself because by causing acute dysentery it may completely exterminate its own race. It is not the ultimate design of the parasite to cause such a destructive lesion but to live in a comparative peace with the

host, establishing a mutual adjustment between them so that it can produce strains which are capable of giving rise to cysts. A high degree of pathogenicity of the parasite is obviously a disadvantage to itself.

After some time, when the effect of the parasite on the host is gradually toned down together with the concomitant increase in the tolerance of the host, the lesions become quiescent and commence to heal. The parasite now finds it difficult to continue its life cycle solely in the trophozoites stage and therefore prepares itself to produce strains which will save the race from extinction. A certain number of these trophozoites are discharged into the lumen of the bowel and are transformed into small precystic forms from which the cysts are developed.

If the parasite happens to enter a resistant host, the injuries produced are minimal (superficial ulcers only). In such a host, E. histolytica not only remains in the trophozoites stage and multiplies at the margin of these superficial ulcers but also discharges from time to time pre-cystic and cystic forms to propagate its species. These persons are thus a constant source of infection to others.

The mature quadrinucleate cysts are the most resistant and infective forms of the parasite and are particularly developed when a state of equilibrium has been established between the host and the parasite. But

the cysts produced in an infected individual are unable to develop in the host in which they are produced and therefore necessitate transference, to another susceptible host where they can grow and continue their life cycle as stated above.

SYMPTOMS AND SIGNS:

It may present as an acute inflammatory process (or) chronic indolent disease. Acute presentations are more common. Rarely, as individual with a ruptured amoebic liver abscess may present in shock.

Interval between amoebic dysentery and development of liver abscess was around 2 months in 50% of patients but it can occur even after few years.

The major complaint is pain usually located in Rt upper quadrant.

Distinguishing clinical characters, in patients with hepatic abscess:

Amoebic	**Pyogenic**
Age <50 yrs	Age > 50 yrs
Male female 10:1	Male Female 1:1
Abdominal pain	Pruritis
Diarrhea	Jaundice
Abdomen tender	Septic shock

Inter costal tenderness	Palpable mass
Hepatomegaly.	

Laboratory diagnosis of Amoebiasis:

The primary aim will be to demonstrate the presence of E. Histolytica in the material obtained from any particular lesion, such as stool, ‘pus’ of liver abscess and sputum.

Diagnosis of Intestinal amoebiasis

A) Symptomatic group cases of acute amoebic dysentery.

Examination of Stool

a) Naked eye (or) macroscopic appearance:

An offensive dark brown semi fluid stool, acid in reaction admixed with blood, mucus and much focal matter is representative of a case of amoebic dysentery.

b) Microscopic appearance:

General microscopically character, under this, attention is given to (I) the character of the cellular exudates and ii) the presence of charcot leyden crystals. The cellular exudates are scanty and consist of only the nuclear masses (Pyknoticbodies), pus cells macrophages and epithelial

cells. The red blood cells are clumped and are reddish yellow (or) yellowish green in colour.

CHARCOT LEYDEN CRYSTALS:

These are also found in other pathological condition of the bowel and therefore not pathognomonic of the stool of amoebic dysentery. Their presence only suggests a careful examination of the stool for E.histolytica. In saline preparation, they appear as diamond shaped (or) whetstone shaped crystal clear and refractile. Their sizes vary from 5 to 50 µm.

C) Demonstration of E.histolytica by examining on unstained preparation microscopically:

Fresh stool, unmixed with any antiseptic or urine should be examined. In acute cases, the amoebic trophozoites can easily be recognized by their characteristic movement and the presence of ingested red blood cells.

2) Examination of blood:

Shows moderate Leukocytosis.

3) Serological test:

In early cases it is always negative because although there is tissue invasion it has not existed long enough to produce detectable antibody.

B) Asymptomatic Group: (Cyst passers or cyst carriers)

(a) Examination of stool:

Demonstration of E.histolytica by:

a) Microscopic Examination of,

(i) A natural stool for cysts (or)

(ii) A smear prepared by concentration method (or)

(iii) A purged stool obtained after a saline cathartic or

(iv)The materials collected by the use of sigmoidoscope. Specimen obtained through the sigmoidoscope yields a positive result only when there are visible lesions in the sigmoidorectal area.

b) Culture examination:

Stools negative microscopically, when cultured, have an various occasion shown the presence of parasites.

c) Animal Inoculation

2) Blood Picture:

It is in no way characteristic.

3) Serological test:

In asymptomatic carries the amoeba present in the stool are in the commensal phase.

DIAGNOSIS OF HEPATIC AMOEBIASIS:

1. Diagnostic aspiration:

   Exploratory puncture is one of the most practical methods for confirming the diagnosis of amoebic liver abscess. The aspirated pus may be examined for the demonstration of tropic forms of E.histolytica.

2. Liver Biopsy:

   E. histolytic may be demonstrated in specimens of liver biopsy taken from cases of miliary amoebic hepatic abscess.

3. Examination of stool:

   Cysts of E.histolytica are present in less than 15% of cases of amoebic liver abscess and gives information regarding the persistence of intestinal infection.

4. Examination of blood:

   Show Leukocytosis, Varying from 15,000 to 30,000 per mm3 of blood. Differential count shows 70-75 percent of neutrophil granulocytes.

5. Serological test:

A positive test indicates the presence of a specific antibody circulating in the blood of infected individual. The various serological tests which may be used as immuno diagnostic methods.

a. Complement fixation test
b. Precipitin test.
c. Immobilization test.
d. Test of Goldman
e. An indirect hemagglutination test.
f. Passive cutaneous anaphylaxis

6) Intradermal test.

7) Radiological Examination:

The right done of diaphragm is generally found to be situated at higher level. Recently hepatic photoscan has been introduced to locate the space occupying lesion in liver.

INVESTIGATIONS FOR LIVER ABSCESS;

Stool examination:

The reported incidence of finding amoebic cyst (or) trophozites in stool of patients with amoebic liver abscess varies considerably.

BALA SEGARAM found amoeba in the stool is less than 15% of ALA patients.

Fresh stool specimen must be preserved in formalin (or) polyvinyl alcohol and can be stained in buffered methylene blue Trichlorane (or) iodine.

KNOGETERED and associates pointed out that a number of substances including antibiotics and antiparasitic drugs, laxatives, antacid, radiological contrast media, enema and antidiarrheal drugs may interfere with stool examination for parasites.

Serological test:

These procedures are necessary to confirm the presence of amoebic liver abscess. It should be obtained as soon as the diagnosis of liver abscess is entertained.

Indirect hemagglutination test (IHA),

Gel diffusion precipitation (GDP),

Complement fixation test,

Latex agglutination test,

Counter current immuno electrophoretic agglutination,

Celluloseacetate precipitation,

ELISA and identification of a recombinant protein is the test available.

If positive, these tests indicate current (or) previous amoebic infection.

The IHA, GDP have been the most frequently used serological test.

IHA will remain positive frequently for many years after invasive amoebiasis, where as the GDP is negative in 6 months, Advantages of GDP are simple to perform, inexpensive and provide information in 24-48 hrs.

The most recently developed test measures 29 KPA peripheral membrane protein of pathogenic E. Histolytica. It differentiates pathogenic form from non pathogenic strains and it is highly specific and reasonably sensitive.

LAB DATA

Leukocytosis is seen in 70% of patients around 50% have elevated alkaline phosphates, Anemia, serum Bilirubin, SGOT, SGPT are mildly elevated. The presence of Jaundice is associated with higher incidence of complication and high mortality rate.

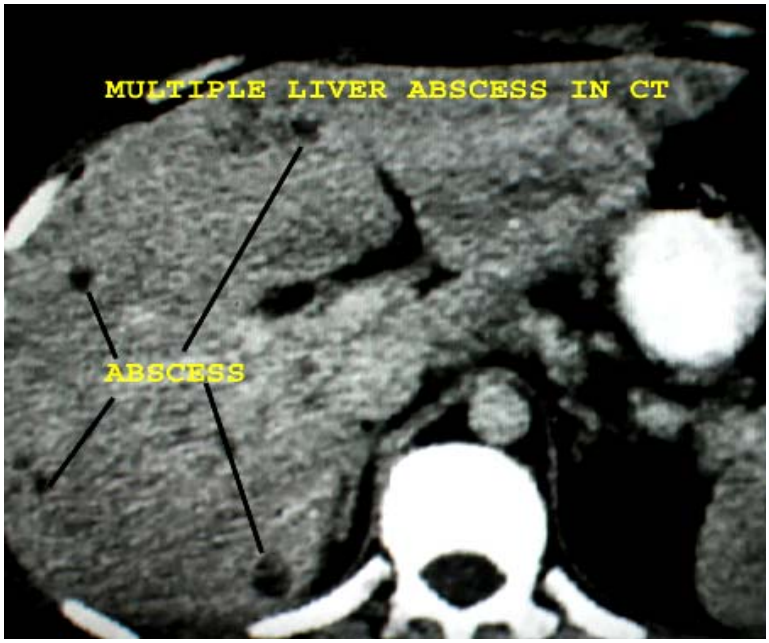
Radiological findings:

About 2/3rds of patients have abnormal chest radiography. The most common radiological finding is elevated right hemi diaphragm, right Pleural effusion, a right lower lobe infiltration and hepatomegaly are found frequently. Fluoroscopy of Diaphragm reveals decreased motion.

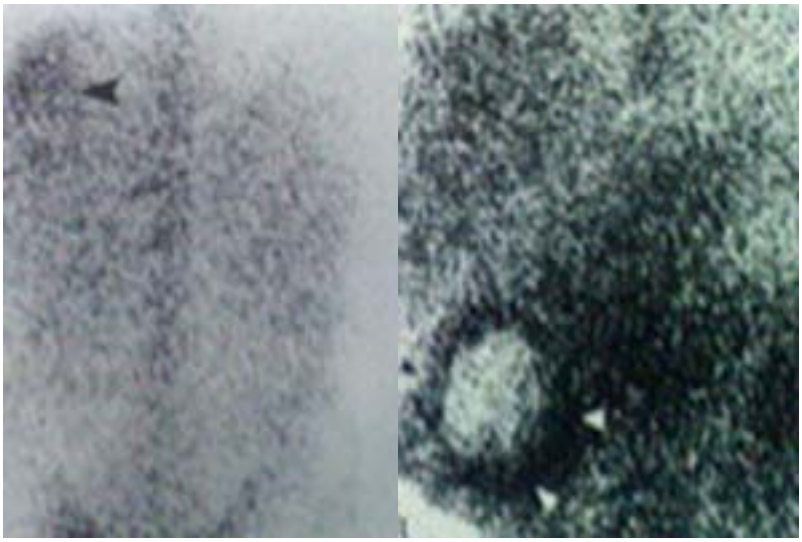
LIVER ABSCESS IN USG

LIVER ABSCESS IN CT

MULTIPLE LIVER ABSCESSES IN CT



GALLIUM SCINTI GRAPHY SHOWING HOTSPOT:



SCINTI SCAN

Gallium scanning is used to differentiate amoebic liver abscess from pyogenic liver abscess. Amoebic will demonstrate a peripheral uptake around a central cold area, where as pyogenic shows increased uptake of gallium throughout the abscess.

Amoebic liver abscess – coldspot.

Pyogenic liver abscess – hotspot in gallium scanning.

USG:

USG helps in determining number, size and location of abscess and can be used as guide for percutaneous aspiration. Ultrasound is non invasive, rapid. It is relatively inexpensive and reproductive. It is also ideal for follow up. Diagnostic accuracy is of 90%.

BOULT BEE and associates reported several sonographic features of amoebic liver abscess.

i) Smooth wall 68%

ii) Internal Echoes were less dense than surrounding normal liver – 84%.

iii) Decreased in echoes of 2/3 (or) more was present in 98%.

CT and Arteriography:

Smaller lesions can be detected, and it is reserved for patients suspicious of having an amoebic liver abscess in which ultrasound is not diagnostic.

Arteriography shows a non vascular occupying lesion. It is invasive and expensive and it may not be as accurate as USG.

MRI:

Amoebic abscess have multiple rims of variable signal intensity. Unfortunately bacterial abscess, Intrahepatic hematoma, and necrotic tumours have similar characteristics. It may be useful in follow up of treated case and also in differentiating if from hepatic neoplasm.

Needle aspiration:

The main reason to aspirate:

1. Diagnostic,
2. To reduce the likelihood of rupture of large abscess,

Diagnostic aspiration is indicated in cases in which the diagnosis remains uncertain despite serological examination and stool examination (or) when bacterial super infection of ALA is suspected.

Needle aspiration should be reserved for situation when.

1. A therapeutic trial with antiamoebic drugs is deemed inappropriate as in case of pregnancy.
2. When fever and pain persists for more than 3 to 5 days after starting appropriate therapy, Aspirations may provide symptomatic relief.
3. In extremely large abscess where rupture is suspected to be imminent especially when pericardial rupture from a left lobe abscess appears likely.

Analysis of Fluid:

It is sterile, both colour and consistency are variable, mostly reddish brown (Anchovy sauce) and it may be white, yellow (or) Green in colour. More reliable characterstic than colour is odour. Amoebic liver abscess is odourless (unless super infection) where as pyogenic liver abscess have foul odour.

Complications:

The incidence of complication varies from 5 to 15%. Most common complication involving pleura and lung, others are rupture into peritoneum, into pericardium, secondary bacterial infection, bacteremia, Amoebiasis cutis etc.

i) Peritoneal and visceral involvements.

Incidence of spontaneous rupture varies between 2.5 to 15% of the cases. Rupture into hollow viscus, a hepatogastric, hepatoduodenal (or) hepatocolonic fistula can occur.

Frequent rupture is uncommon, occurs in nutritionally depleted and moribund patient. Sudden bloody discharge, hematemesis may occur in colonic (or) gastric fistula.

USG and CT show peripheral fluid collections in case of amoebic liver abscess.

Indications for Laparotomy:

1. Doubtful diagnosis
2. Concomitant hollow viscus perforation with fistula formation.
3. If conservative treatment fails.

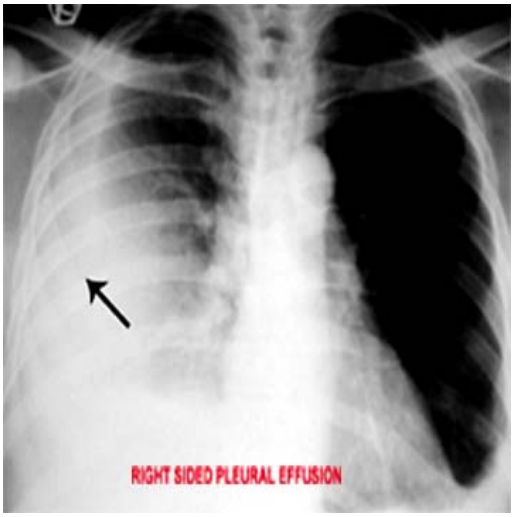
After Laparotomy, tube drains are inserted and retained as necessary Hollow viscus perforation must be dealt with on their own merits with sterilization, proximal diversion, serosal patch closure.

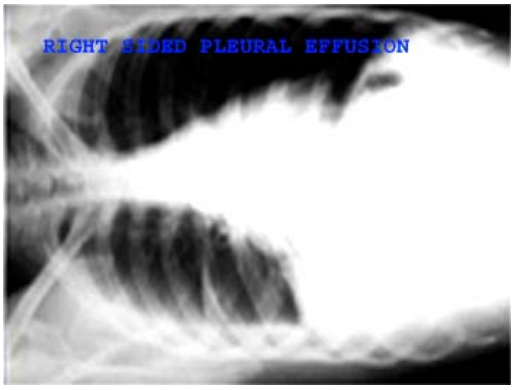
Postoperative management:

Antiamoebic therapy in the form of IV metronidazole is combined with broad spectrum antibiotics.

Thoracic and plueropulmonary involvement:

It may be due to sympathetic effusion (or) rupture into pleural cavity. Patient may manifest with dyspnea and dry cough (or) right hypochondrial pain, Basal crepitations and pleural rub. USG and CT often pick up the pleural effusion. Radiography reveals a homogeneous opacification through right hemi thorax with displacement of mediastinum to the opposite side.


RIGHT SIDED PLEURAL EFFUSION


RIGHT SIDED PLEURAL EFFUSION


RIGHT SIDED PLEURAL EFFUSION

Treatment:

It consists of thoracocutaneous ICD. Rupture of the abscess into bronchi may present with chocolate coloured sputum.

Pericardial Involvement

Abscess of left lower lobe of liver are more prone to pericardial complication which may range from asymptomatic pericardial effusion to cardiac tamponade.

In the presence of cardiac tamponade, aspiration of pericardium must be performed along with drainage of liver abscess followed by anti amoebiasis treatment.

Although investigation agrees that all patients should be treated with amoebicidal agents some controversies persist with regard to choice of medication and the need to aspiration (or) surgical drainage.

Amoebicidal Drugs:

In 1966 POWELL and colleagues introduced metronidazole. It is highly effective and less toxic as well as being effective against intestinal and hepatic amoebic organisms.

An oral course of metronidazole 750mg 3 times a day for 10 days cures approximately 95% of patients with amoebic liver abscess.

Other agents currently under investigation are Tinidazole, Niridazole and secnidazole. Emetine and dehydroemetine are indicated primarily when patient develop pulmonary complication. These drugs eliminate invasive organisms. Luminal amoebicides are,

1. Diloxanide furonate,
2. Clioguinal,
3. Tetracycline,

Secnidazole 1.5gm / day – 5days

Tinidazole 500 mg bd for 5- 10 days.

A course of chloroquine is given to,

1. Decrease the intestinal reservoir.
2. Ensure no motile forms in liver.

Needle aspiration

Indication:-

1. Persistence of symptoms despite adequate drugs > 72 hrs.
2. Concern regarding rupture abscess > 250ml in volume at Lt lobe.
3. Secondarily infected abscess
4. Large abscess.

Those when urges against routine aspiration, say resolution time was unchanged by therapeutic aspiration and it increase the secondary infection.

Those who are in favour of aspiration claims that the advantage of preventing rupture and relieving of pain out weigh the low incidence of secondary infection.

Aspiration also appears valuable when metronidazole therapy is contra indicated, such as during pregnancy.

Percutaneous catheter drainage (PCD):

The reason why PCD has not become popular include, fear of bacterial super infection and the fact that most of the patients respond to amoebicidal drugs very well with or with out closed aspiration.

BALASEGARAN recommended the use of soft drainage catheter of sufficient size to adequately drain the thick viscous contents of amoebic liver abscess.

In 1992 **Flice and his Italian colleagues** demonstrated the advantages of PCD which relives symptoms early and resolves the abscess cavity earlier. They also documented the use of intraleision amoebicidal therapy.

Open surgical drainage:

It is reserved for the patients with complications.

1. Abscess that have failed to respond to more conservative therapy.

2. in the rare event of life threatening hemorrhage.

3. Abscess that erodes into neighboring viscus.

4. Patient with septicemia from secondary infected amoebic abscess.

5. Where the abscess is inaccessible to any closed technique**.**

PYOGENIC LIVER ABSCESS (PLA)

Patients with Pyogenic liver abscess are more likely to be older, to be female and of biliary etiology.

In 1826 JOHN BRIGHT provides the first description of the disease.

In 1938 OSCHER and associates made out that appendicitis was the common site of origin.

Etiology and Pathogenesis:

Most PLA are due to infection in the biliary (or) intestinal tracts.

Routes of infection:

1) Biliary

2) Portal vein

3) Hepatic artery

4) Direct extension

5) Traumatic

6) Cryptogenic.

Biliary system is common origin of PLA 55%. Frequent sources of portal vein sepsis resulting in liver abscess include diverticulitis, perforated ulcer and carcinoma. Appendicitis, pelvic and pancreatic abscess are relative rare sources.

Mc Donald and Howard noted that 65% are in right lobe 12% in left lobe and 23% bilateral.

The percentage of multiple abscesses varies between 30 and 70% and mortality closely correlates with the number of abscess. Gram (-) ve aerobes were cultured in 2/3 of patient and gram +ve aerobes were found in around 28% of patients. PLA is sterile in about 7% of patients.

Factors associated with liver abscess after biliary tract surgery.

1) Anastomotic stricture → Benign
   → Malignant
2) Foreign body, suture, stent, stump syndrome.
3) Intrahepatic bile duct stricture.
4) Common duct stones.
5) Intra hepatic stones.
6) Vascular injury.
7) Altered G.I. flora, achlorhydria, dueodenal diverticula.

Organism:

The pyogenic nature of PLA is usually confirmed by micro biological culture. Positive abscess cultures are found approximately in 80% whereas blood cultures are positive in approximately in 60% of cases.

E-Coli is frequent organism. Others include Klebsiella, enterococci, Pseudomonas, citrobacter, Proteus, strepto and staphylococcus.

Hepatic abscess following trauma caused by staph, streptococcus as other aerobic gram –ve organism.

In children with chronic granulomatous disease, hematological malignancies, staph aureus is the predominant organism causing liver abscess.

Few patients with AIDS presenting with abscess, mycobacterium tuberculosis is common infecting organism.

Symptoms and sign:

Most of patients present with acute symptoms. Fever is the most common presenting symptom others are right upper quadrant pain, malaise and nausea.

The most common physical sign are enlarged tender liver in 60% of cases and Jaundice in 30% of cases

INVESTIGATIONS:

Laboratory data:

Leukocytosis and anemia observed in 2/3rd of patients, elevated alkalinephosphatase and Gamma glutamyl transpeptidase in 96% of patients. Hyperbilirubinemia and hypoalbuminemia associated with risk.

Radiological findings:

X-ray chest:

Elevated right hemi diaphragm, right lower lobe atelectasis, right pleural effusion.

X-ray abdomen erect:

Hepatomegaly,

Gas with in the abscess.

Cholangiography:

As the incidence of ascending cholangitis in case of PLA has increased, cholangiography has become an important aid in the diagnosis.

Liver scan:

Helps in early detection of large abscess, not useful in detecting smaller abscess <2cm. Differentiation between abscess and tumor is not possible with technetium and gallium, but Indium (^{III} In) scans may be of some help.

USG:

Sensitivity is reported up to 85% to 95% since liver dome cannot be visualized always. Fatty infiltration may produce markedly echogenic liver and multiple abscess are difficult to detect.

Has same resolution as SCINTI scan but has no radiation. It can identify only the abscess of more than 2 cm in diameter. It is useful in differentiating the cystic and solid abscess and in diagnosing the gall stones.

CT:

Sensitivity is up to 95%. It detects intra hepatic lesion up to 0.5cm. CT better delineates small abscess near the diaphragm and abscess in fatty liver. But it cannot always differentiate the abscess from other SOL.

MRI:

Micro abscess which is as small as 3 mm, in diameter can be identified. Its advantages over CT are that it gives useful information about hepatic venous anatomy, from which it gives important guidelines in patients who require liver resection.

Angiography:

Reserved for patients in whom CT and MRI have not provided a definite diagnosis.

Bacteriology:

Gram negative enteric bacteria include E-Coli, Klebsiella, enterobacter, Proteus species are isolated from 50% to 75% of liver abscess.

Anaerobic bacteria about 40% are cultured from PLA. Gram +ve aerobes are about 30% of abscess.

Polymicrobial abscess occurs most commonly in adults and are typically solitary.

FUNGAL HEPATIC ABSCESS

Usually arise in patients with leukemia, Lymphoma and in patients with other malignancies receiving chemotherapy.

Complication:

Complication occurs in 45% of patient associated with significant higher mortality, Pulmonary, pleural complication, septicemia, subphrenic (or) subhepatic abscess rupture into peritoneum, pericardium and multi organ failure.

Treatment:

Antibiotics:

Early institution of antibiotic therapy is important. The antibiotic regimen is based on knowledge of spectrum of organism isolated in PLA.

1) Gram –ve aerobes.
2) Streptococcal species
3) Anaerobes

Unless a specific bacteria have been isolated. Combination of penicillin, an aminoglycoside, an agent against bacteriodes fragilis will be necessary. The length of antibiotic therapy should be individualized. Patient with multiple military abscess require 4 to 6 weeks of therapy. Where as small solitary abscess required a shorter course.

It is useful in multiple small abscesses that are not associated with abdominal disease that requires surgery.

Needle aspiration:

It is useful in young, healthy patients having no other source of intra abdominal infection.

Percutaneous drainage (PCD):

This should be done along with antibiotics.

PCD should not be used in,

1) Associated disease that requires open surgery
2) Coagulopathy
3) Anatomical inaccessibility
4) multiple small abscess
5) Ascites

Open drainage:

It is indicated in PLA with intra abdominal disease requires surgery and in areas inaccessible to PCD (i.e.) close to portal vein. It may be through extra peritoneal (or) transperitoneal approach.

Extra peritoneal approach:

This is recommended because of fears of contamination of pleural (or) peritoneal cavity with transplueral (or) transperitoneal approach.

Here high abscess cavity is entered through areas of adhesion between liver and parietal peritoneum. The exact route was dictated by the position of abscess and it should be as short as possible.

Disadvantages:

Disadvantages of this approach are that it does not allow adequate exploration of the entire liver (or) for recognizing of an inadequately treated intra abdominal sources of infection.

Transperitoneal approach:

This can be performed safely now with systemic antibiotic coverage and proper surgical technique

1. Excellent Exposure of entire liver.
2. Best drainage site can be determined.
3. Multiple abscess can be located ,
4. If the source of sepsis is occult exploration of entire abdomen can be done.
5. If indicated CBD exploration and drainage can be done.

Once the abdomen has been explored the primary focus of infection has been managed appropriately and liver should be palpated carefully. If the abscess is not obvious at surgery, Intra operative USG is used.

Liver resection is occasionally required for patients with pyogenic liver abscess. The indication for this is usually hepatolithiasis (or) Intra hepatic biliary stricture. In severe hepatic disease manipulation may produce life threatening bacteremia.

Laparoscopic drainage is an effective alternate for patients requiring open surgical drainage. The advantages are reduced analgesic requirements, reduced morbidity, faster postoperative recovery and shorter hospital stay.

Results of Treatment:

Mortality rate varies from 24% to as high as 85% and for multiple abscesses it is extremely high around 95%. Solitary amoebic abscess have better results.

AIMS OF STUDY:

1. To study the various clinical presentation and their significance in diagnosis of liver abscess.
2. To form a diagnostic work up for the diagnosis of liver abscess.
3. To find its appropriate indication for various modalities of treatment in amoebic liver abscess.
4. To find out the most appropriate and cost effective approach in the management of amoebic liver abscess.

Materials and methods:

Hundred six cases of liver abscess admitted in THANJAVUR MEDICAL COLLEGE AND HOSPITAL, between August 2006 and July 2008 was studied.

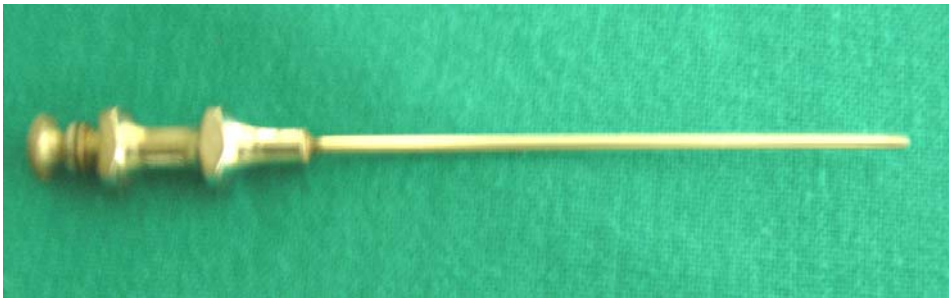
All the patients were thoroughly examined and case sheets were written in the same set patterns to facilitate latest comparison.

All of them had several investigation required to approach the diagnosis and there were diagnosed as ALA (or) pyogenic liver abscess.

They were subjected to various form of treatment available to find our appropriate indications.

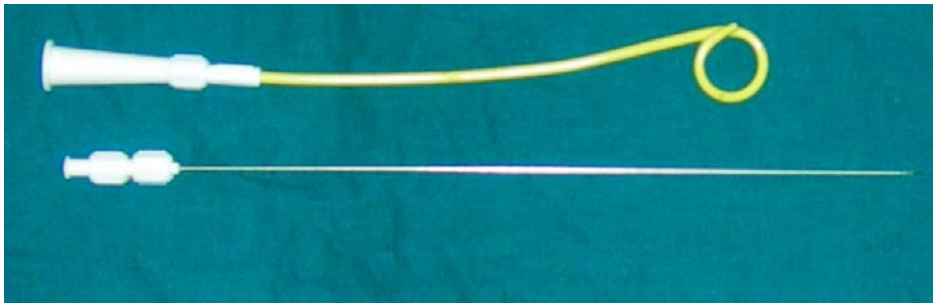
PERCUTANEOUS LIVER ABSCESS ASPIRATION NEEDLE

(16G SIZE)



PERCUTANEOUS LIVER ABSCESS DRAINAGE CATHETER

(14F SIZE)



Results and resolution time of all cases were recorded and compared to find out an appropriate and cost effective approach for a give case.

Among 93 ALA, 90 Patients regularly attended follow up.

Among these, 90 Patients were selected and divided into 3 groups in random means (A,B,C,)

Now each group has is patients

Group A - Treated with drug alone.

Group B - with drug and therapeutic aspiration

Group C - with drug and percutaneous catheter drainage.

Adopted Therapeutic Protocol

1. Medical

Tab. metrogyl 750mg Tds for 10 days

2. Needle aspiration:-

16G (or) 18 G Aspiration needle (or) 3 way adopter is used as single pride under USG guidance.

1. First aspiration followed by drugs
2. If symptoms not decreasing after 3 days, Repeat USG done and the cavity size assessed.
3. If the cavity is increasing in size (or) not decreasing.

2nd aspiration should be done and drug therapy was continued. Still the symptoms are not subsided by the 7th Post aspiration day and USG showed the cavity not decreasing (or) increasing in size, consider PCD.

PERCUTANEOUS NEEDLE ASPIRATION

3. Percutaneous drainage: (PCD)

By using 14f Multipurpose pig tail drainage catheter.

Under USG guidance PCD is being done with close drainage system.

Record symptoms:

1. Increased, decreased (or) remains same,
2. Quantity of drainage in ml,

3. Type of fluid purulent / Necrotic liver tissue. (anchovy sauce /serous)

Removal of PCD:

1. Quantity less than 30 ml / 8 hrs

2. Not purulent.

3. When patient is free of symptoms, patient is fit for removal of PCD. Repeat USG evaluation and arteriogram to assess cavity size. Note down the decrease in size of cavity and PCD can be removed.

Other wise if it is purulent, the patient is with evidence of secondary infection, do pus culture and sensitivity. Start broad spectrum antibiotic until results arrive .Then treat the patients according to it.

Complication of PCD:

1. Peritonitis,

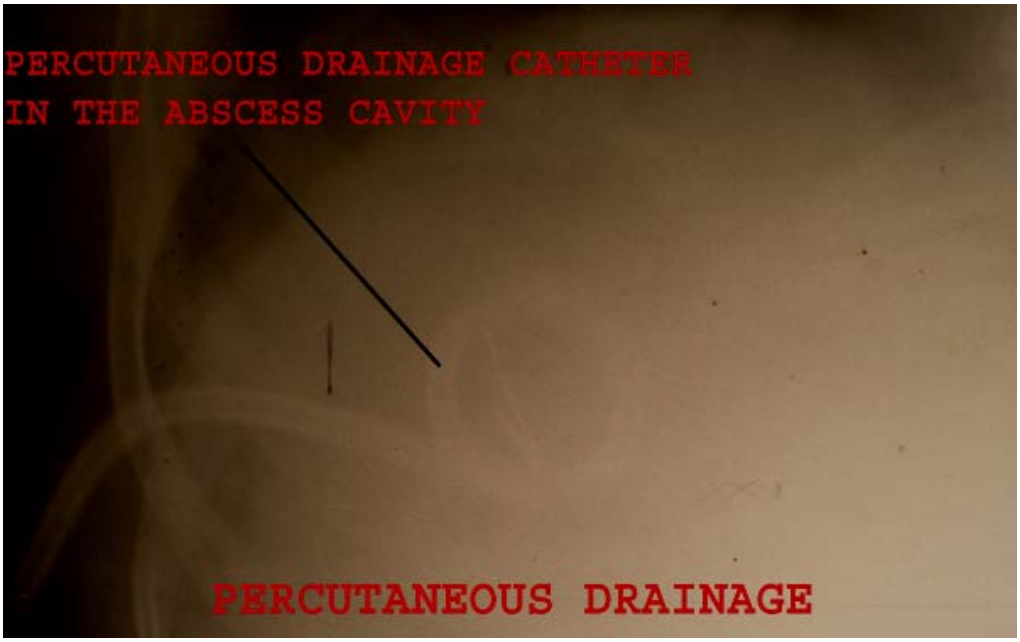
2. Rupture in to pleural cavity.

PERCUTANEOUS DRAINAGE

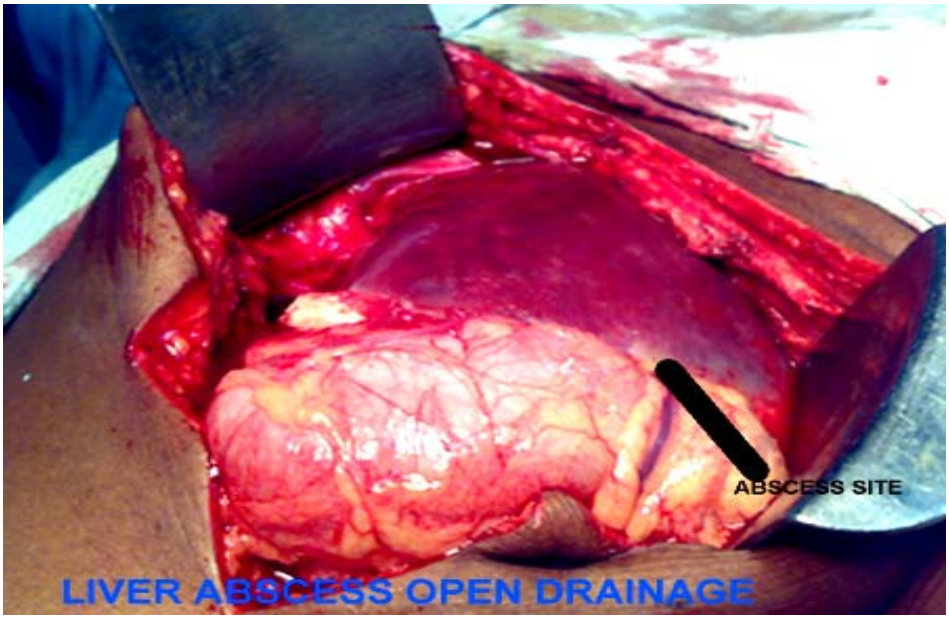
PERCUTANEOUS DRAINAGE

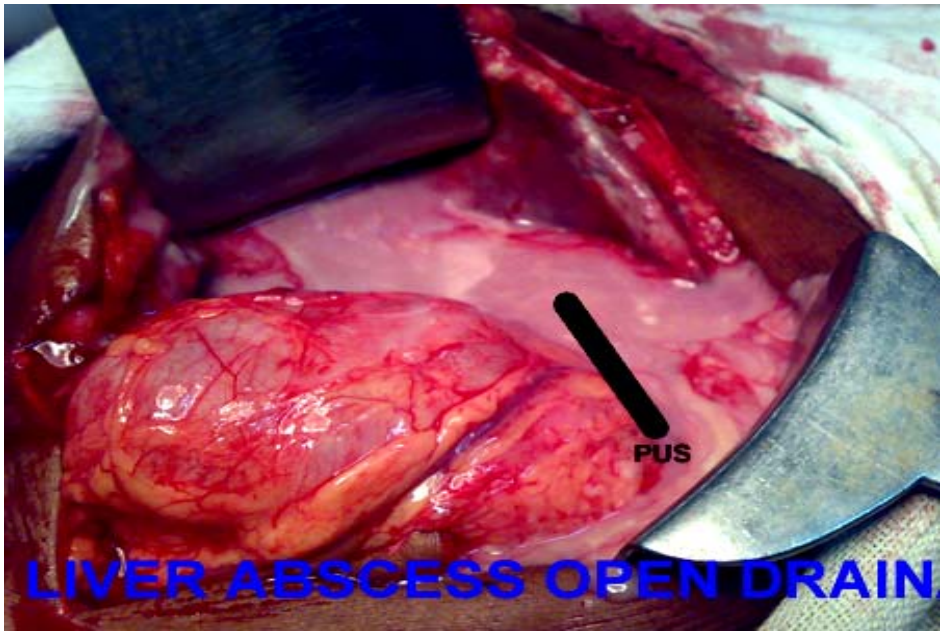
PERCUTANEOUS CATHETER DRAINAGE

PERCUTANEOUS CATHETER DRAINAGE



OPEN DRAINAGE





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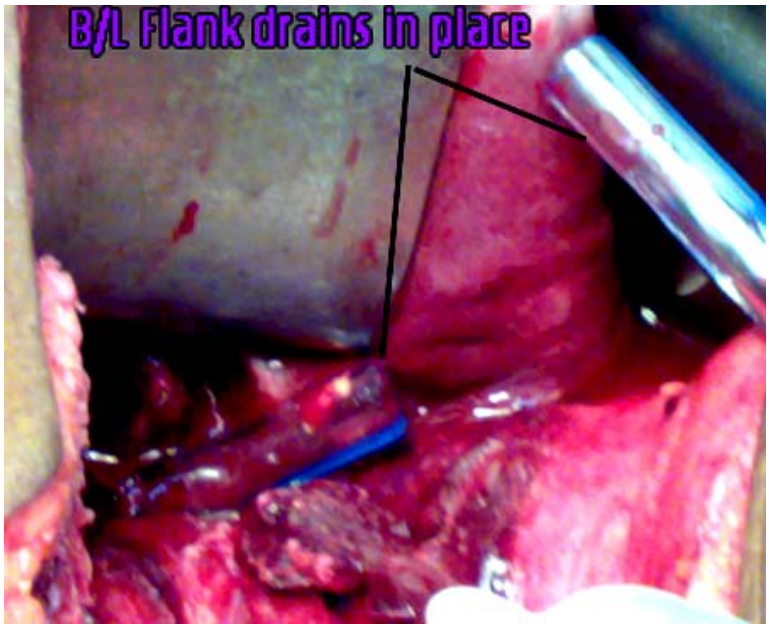
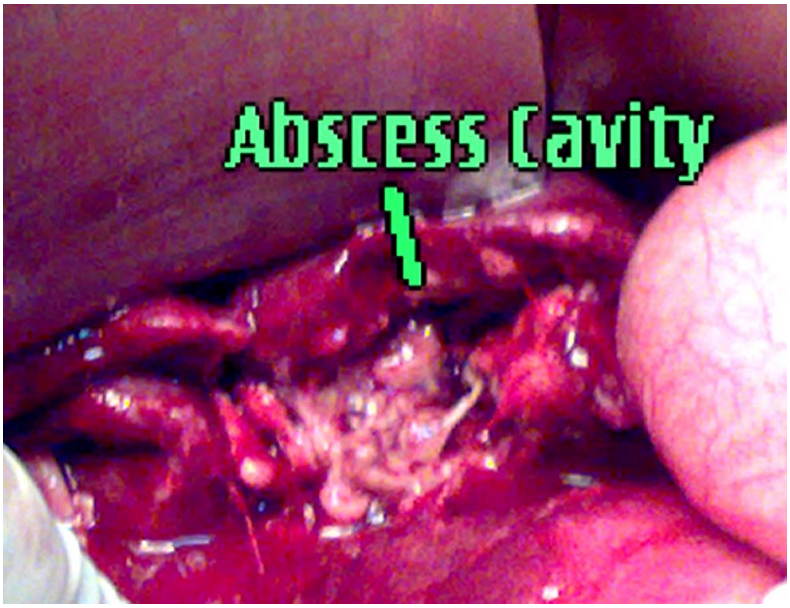




ANALYSIS OF DATA

Total patients studied - 106

Amoebic liver abscess (ALA) - 93

(Three Patients of Amoebic liver abscess Treated with Drugs alone

Didn't come for follow up)

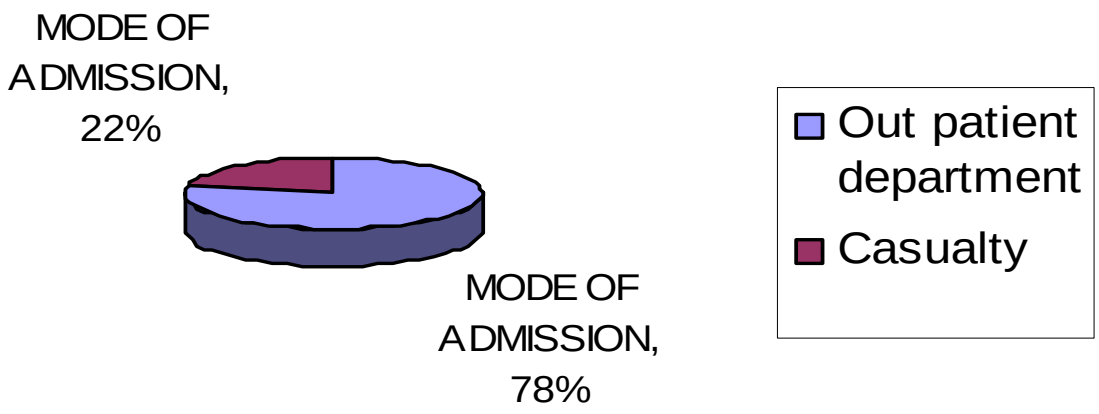
Pyogenic liver abscess (PLA) -13

Table: 1

Mode of admission:

Mode of admission:	Out patient department	Casualty
ALA	77	16
PLA	6	7

MODE OF ADMISSION



GEOGRAPHICAL DISTRIBUTION

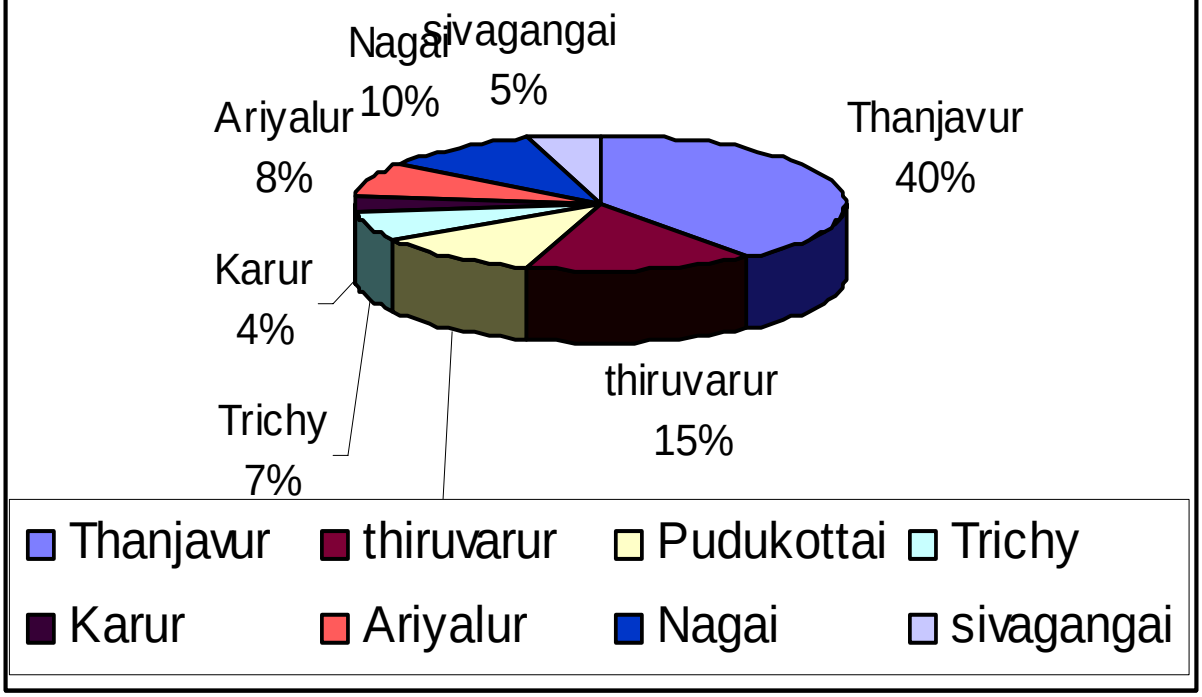


Table: 2

Geographical distribution:

Geographical Area	Thanjavur	thiruvarur	Pudukottai	Trichy	Karur	Ariyalur	Nagai	sivagangai
No of patients	42	16	12	7	4	9	11	5

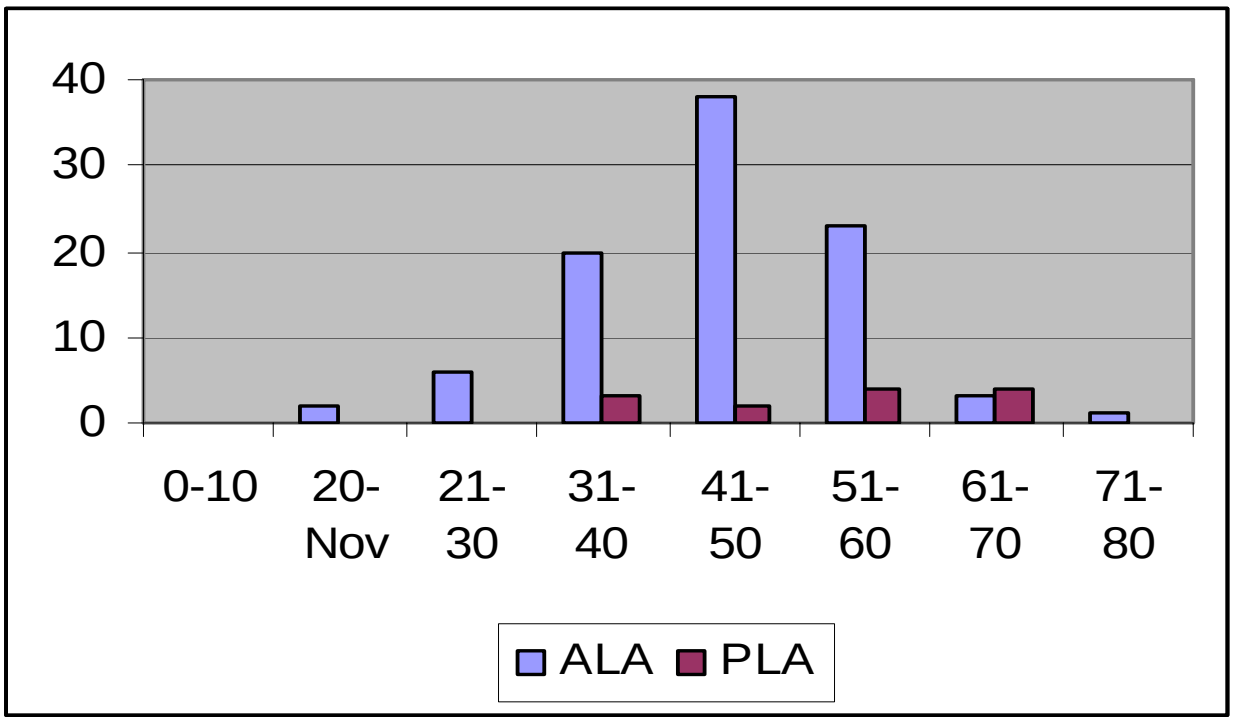
Table: 3

AGE INCIDENCE:

Age	ALA	%	PLA	%
1-10	-	-	-	-
11-20	2	2.1%	-	-
21-30	6	6.5%	-	-
31-40	20	21.5%	3	23.1%
41-50	38	40.9%	2	15.7%
51-60	23	24.7%	4	30.8%
61-70	3	3.2%	4	30.8%
71-80	1	1.1%	-	-

Total	**93**		**13**	

BAR DIAGRAM: AGE INCIDENCE



BAR DIAGRAM: SEX INCIDENCE

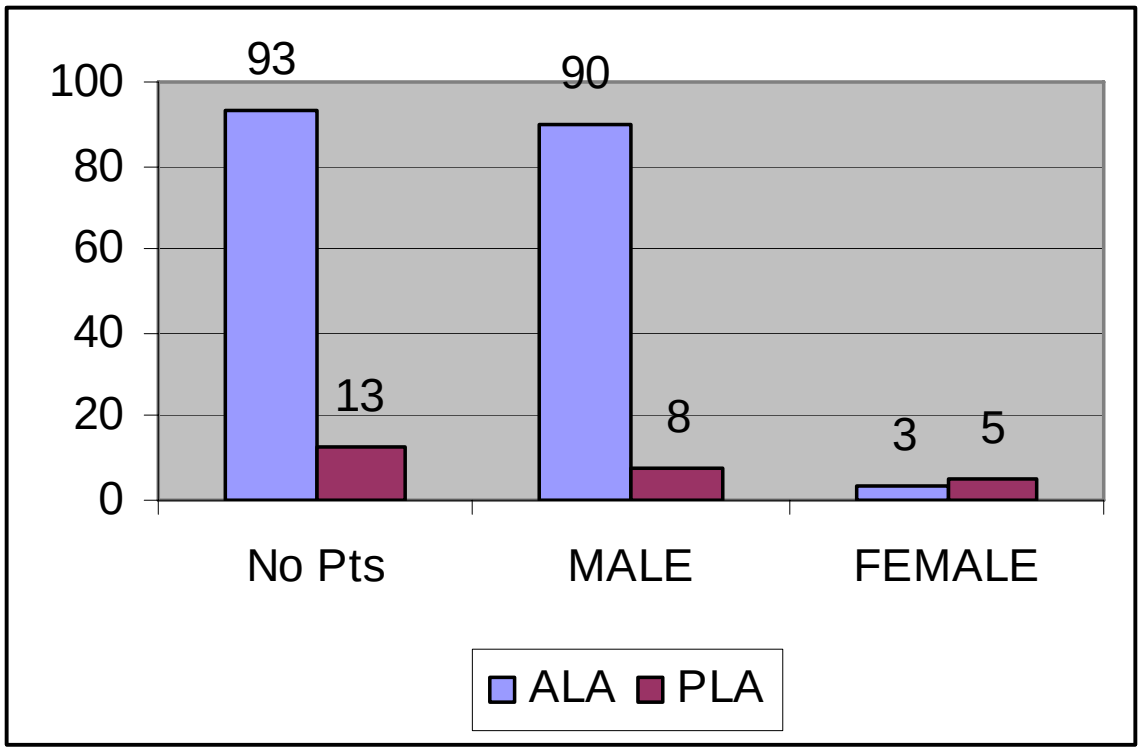


Table: 4

SEX INCIDENCE:

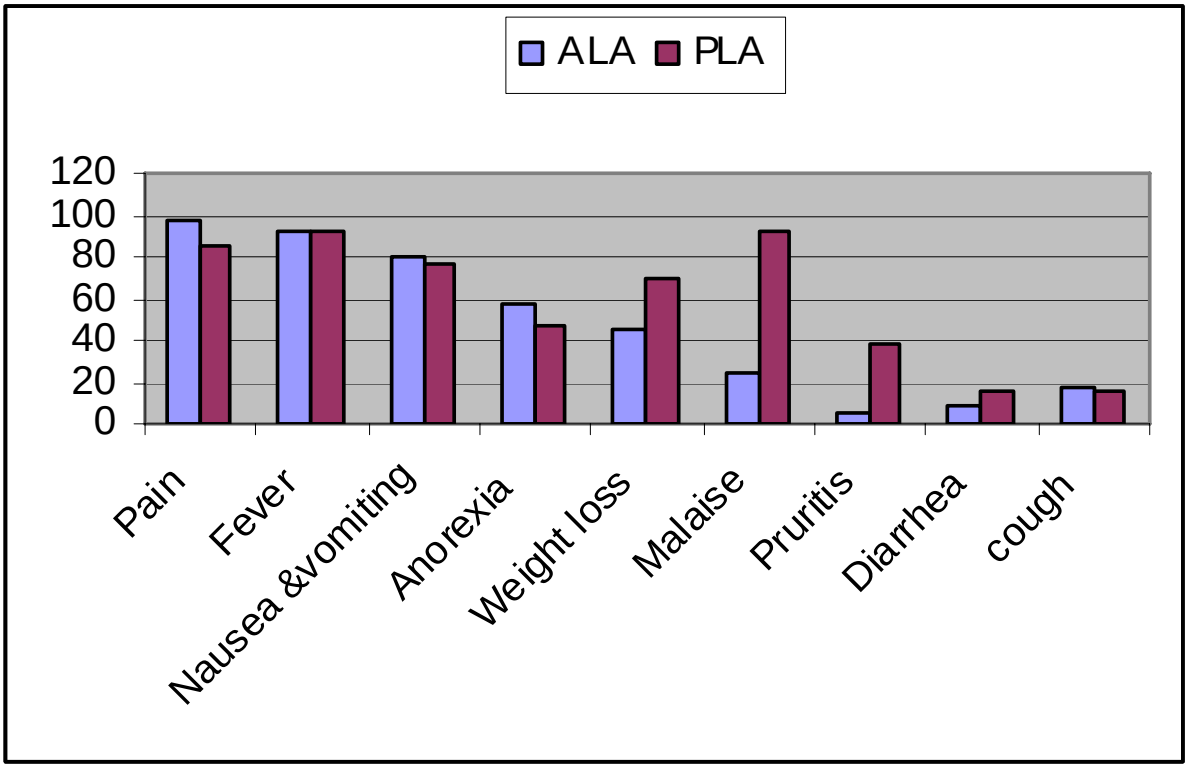
Type	No.of Patients	Male	%	Female	%	M:F Ratio
ALA	93	90	96.8%	3	3.2%	10:1
PLA	13	8	61.5%	5	48.5%	1.6:1

Table 5

SYMPTOMS

No.	SYMPTOMS	ALA		PLA	
		No	%	NO	%
1	Pain	90	96.8	11	84.6
2.	Fever	85	91.4	12	92.3
3.	Nausea &vomiting	74	79.6	10	77.0
4.	Anorexia	54	58.1	6	46.2
5.	Weight loss	42	45.2	9	69.2
6.	Malaise	22	23.6	12	92.3
7	Pruritis	5	5.4	5	38.5
8	Diarrhea	8	8.6	2	15.4
9	cough	16	17.2	2	15.4

BAR DIAGRAM: SYMPTOMS IN LIVER ABSCESS



BAR DIAGRAM: SIGNS IN LIVER ABSCESS

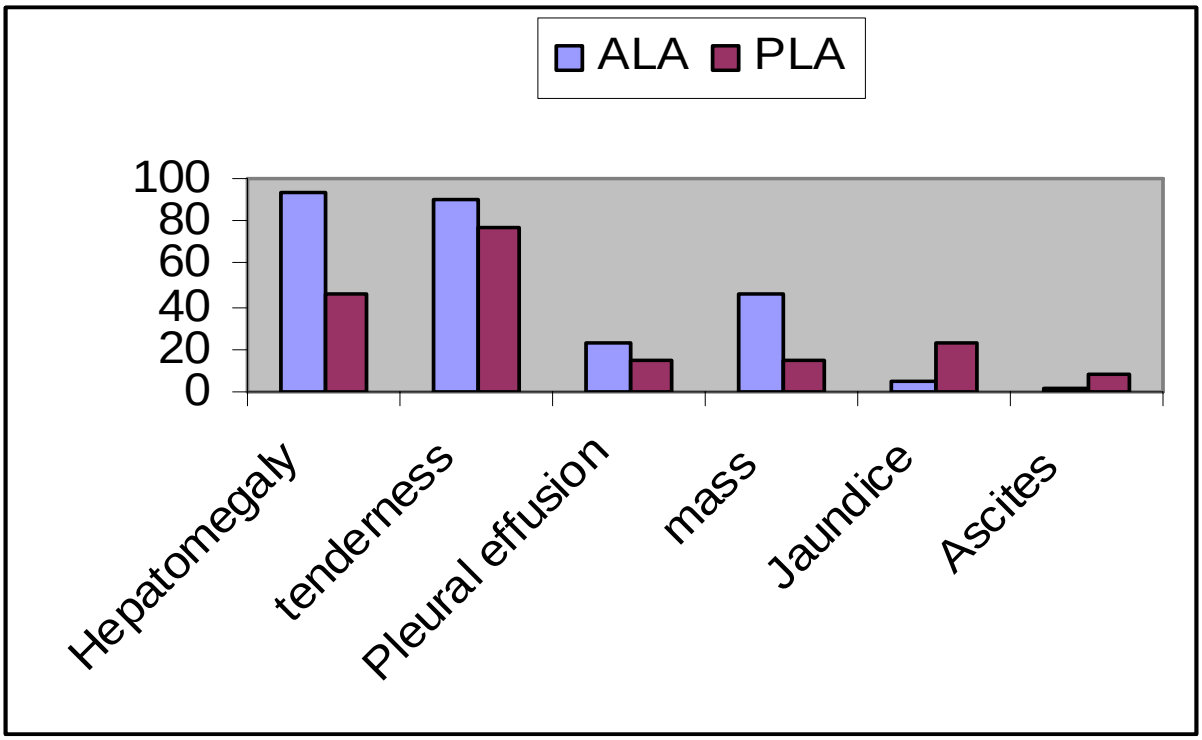


Table 6

Signs:

No	Signs	ALANo. of Patients	%	PLANo. of Patients	%
1.	Hepatomegaly	87	93.5	6	46.2
2.	Right Upper Quadrant tenderness	84	90.3	10	76.9
3.	Pleural effusion	22	23.7	2	15.4
4.	Right upper quadrant mass	42	45.2	2	15.4
5.	Jaundice	4	4.3	3	23.0
6	Ascites	1	1.1	1	7.7

Table 7

Comparison of Lab findings:

No	Symptoms	ALA No. of Patient	%	PLA No. of Patient	%
1.	Anemia	30	32.3	10	76.9
2.	Leukocytosis	80	86.0	13	100

3.	↑ Serumalkaline Phosphatase	13	14.0	13	100
4.	↓ Sr.Albumin	40	43.0	12	92.3

TYPE OF ABSCESS IN USG;

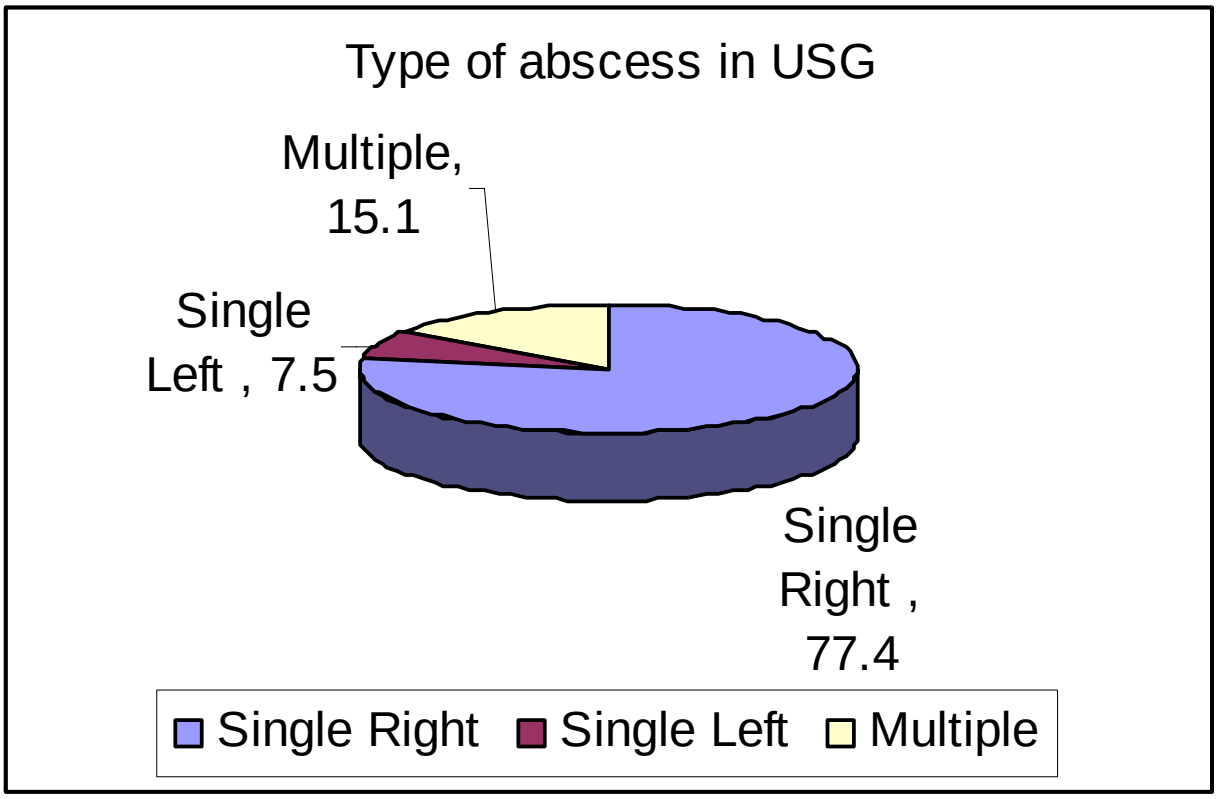


Table 8

USG FINDINGS

No	Type of Abscess	No of Pts	%
1.	Single Right	82	77.4
2.	Single Left	8	7.5
3.	Multiple	16	15.1

Table 9

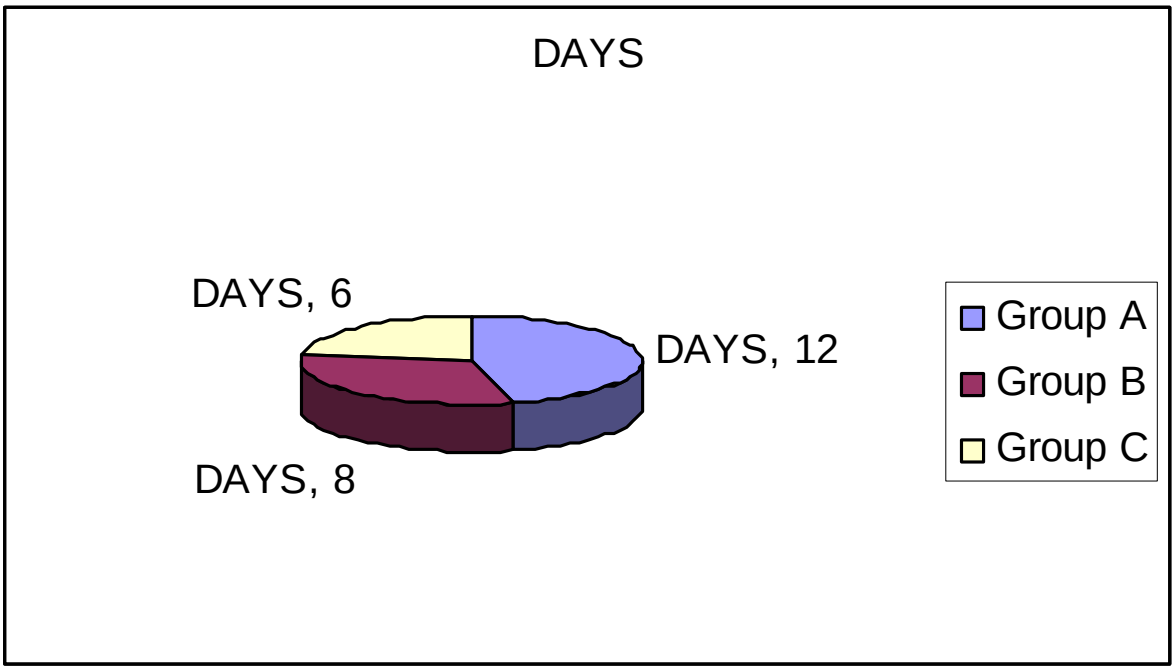
Comparison of results of the study group in ALA,

Relief of Symptoms and Resolution time:

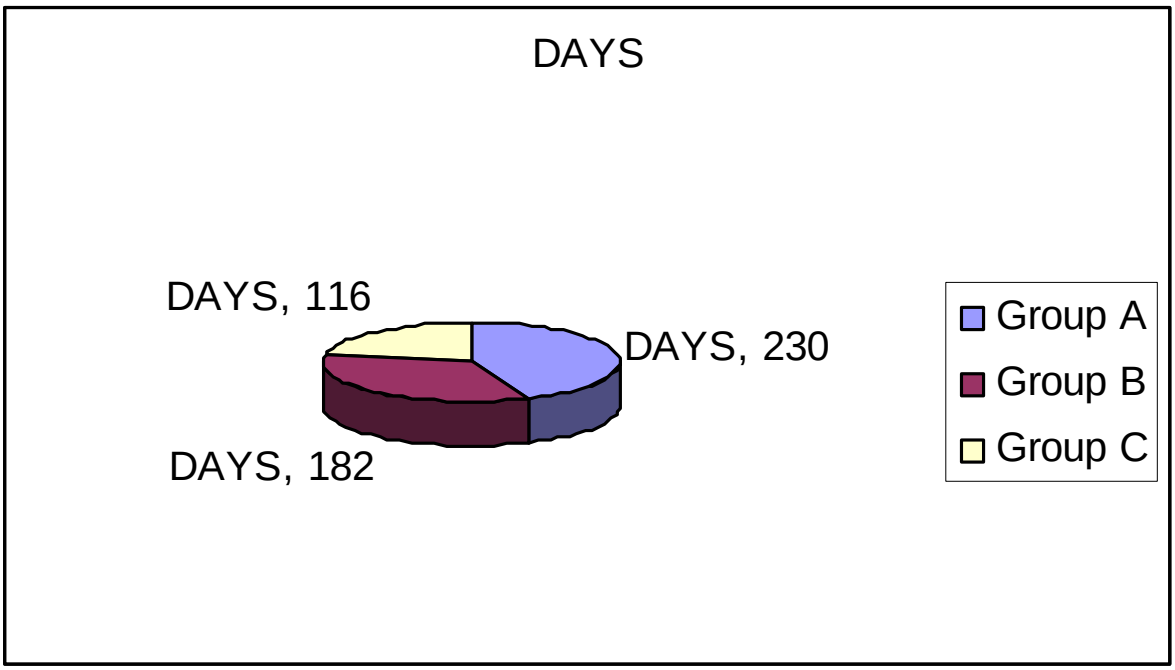
No	Study Group	Relief of Symptoms	Resolution time
1.	Group A Drug Alone	12 days	230 days

2.	Group B Drug + needle Aspiration.	8 days	182 days
3.	Group C Drug + PCD	6 days	116 days

Comparison of relief of symptoms in days:



Comparison of Resolution time in days:



DISCUSSION

The study was conducted in Thanjavur Medical College and Hospital between August 2006 and July 2008.

106 Patients were admitted during the above period and all the patients were thoroughly examined and case sheets were written in the same set patterns to facilitate latest comparison.

All of them had several investigation required to approach the diagnosis and they were subjected to various form of treatment available to find out appropriate indications.

The results are recorded as follows:

Among 106 patients, 93 were diagnosed as ALA, 90 patients had attended regular follow up:

Among these 90 patients were selected and divided into 3 groups in random means.

Each group of patients, were treated as per protocol.

In this study most of the patients belong to third to sixth decade and also shows male predominance (Male: Female 10:1)

Most of the patients were admitted through out patient department with complaints of pain and fever. Other symptoms include Nausea, Vomiting, and Anorexia, Weight loss, malaise, pruritis, Diarrhea and cough.

Hepatomegaly and Right upper quadrant tenderness remains most common sign in majority of cases. Other signs include Right upper quadrant mass, pleural effusion, jaundice, Ascites.

Eighty six percentages of patients had Leukocytosis and more than ninety percentages of patients had solitary right lobe abscess in radiological examination.

The study shows that patients treated with drugs and percutaneous drainage had earlier relief of symptoms with minimal resolution time.

Four patients were treated with open drainage, among them one patient presented with ruptured liver abscess, other three patients were treated with open drainage for the indication of inaccessible for closed drainage.

CONCLUSION:

1. **Age incidence. ALA is common in 3rd, 4th and 5th decades; PLA is common in 5th and 6 th decade.**
2. **In this study the male to female ratio in**

   ALA → 10:1

   PLA → 1.6:1

Most of the patients are labourers and of low socio economic group.

It is more common in alcoholic.

3. **Most of the patients present with right hypochondriac pain in ALA and high grade fever in PLA.**

The common clinical symptoms:

Sl.No.	ALA	%	PLA	%
1.	Rt. Hypochondrial pain	96.8%	High grade fever	92.3%
2.	Low grade fever	91.4%	Pain abdomen	84.6%
3.	Nausea and vomiting	79.6%	Wt. loss	69.2%
4.	Anorexia	58.1%	Malaise	92.3%

The common clinical signs:

Sl.No.	ALA	%	PLA	%
1.	Hepatomegaly	93.5%	Rt. Hypochondrial tenderness	76.9%
2.	Rt. Hypochondrial tenderness	90.3%	Hepatomegaly	46.2%
3.	Pleural effusion	23.7%	Jaundice	15.4%

4. **Important Laboratory finding are elevated serum alkaline phosphatase, Leukocytosis, Anemia and decreased serum albumin.**

5. **X-ray chest shows elevated Rt.hemidiaphragm**
6. **USG – mostly present as large, single abscess cavity.**
7. **Though ALA is treated mainly by drug therapy, radiological and surgical interventions are still warranted in few instances.**
8. **In ALA the relief of symptoms in days**
    a) **With drug alone 12 days.**
    b) **Drugs with Needle aspiration – 8 days.**
    c) **PCD with drugs – 6 days.**

9. **In ALA the average resolution time in**

    a) **With drug alone 230 days.**

    b) **Drugs with Needle aspiration – 182 days.**

    c) **PCD with drugs – 116 days.**

PCD IS MOST ADVANTAGEOUS, BECAUSE OF EARLY SYMPTOM RELIEF AND LESS RESOLUTION TIME.

10. **The incidence and complication following PCD and therapeutic aspiration is not increased.**
11. **Four cases were treated with open surgical drainage procedure for an appropriate indication; still there is role for open drainage in case of complicated liver abscess.**

Annexure I

PROFORMA

Case No : **IP No.**
Name : **Occupation:** **D.O.A.**
Age : **Religion :** **D.O.D.**
Sex :

1. **Complaints of the patient**
2. **History of presenting illness.**
3. **Pain abdomen present (or) absent.**

A.

1. **Onset**
2. **Duration**
3. **Type: Dull aching / sharp/ discomfort.**
4. **Radiation → Present**
   → Absent
5. **Relation to Cough → Increase**
   → Decreased
   → No change
6. **Relation to Food → Increased**
   → Decreased

B. Fever → Present → High-grade → Sustained Spike
→ Low-grade
→ Absent

C. Nausea & Vomiting

D. Appetite → Increased
→ Decreased
→ No change

E. Loss of Weight → Present
→ Absent

F. Jaundice → **Present**
→ **Absent**

G. Diarrhea → **Duration**
→ **Frequency**
→ **Nature** → **Mucous**
→ **Bloody**
→ **Quantity**

H. Cough → **Duration**
→ **Dry (or) Productive**

I. Pruritis → **Present**
→ **Absent**

J. Lump → **Present**
→ **Absent**

→ **Site**
→ **Duration**
→ **Progress**

3. H/o. Previous Illness

1. Similar previous complaints.

2. H/o. Diarrhoea → **Present**
→ **Absent**

3. H/o previous Treatment if any

4. How many weeks of Hospital Stay.

4. Personal History

Diet → **Vegetarian**
→

Mixed

Smoking

Menstrual History

Alcoholic

5. Family History → **Present**, **Absent**

6. Socio Economic Status → **Low**, **Middle**, **High**

7. Physical Signs

8. General Examination

1) **Built and Nourishment**
2) **Anemia**
3) **Jaundice**
4) **Skin Lesion**
5) **Lymphadenopathy**
6) **Temperature**
7) **Respiration**
8) **Pulse**
9) **BP**
10) **Pedal edema**
11) **Any Other**

Local

Abdominal Examination

a. Inspection: Moves with Respiration → **Normal**, **Restricted**

Absent

Mass → **Present**
→ **Absent**

Distension → **Present**
→ **Absent**

b. Palpation

Tenderness → **Present**
→ **Absent**

Hepatomegaly → **Present** → **Soft**
→ **Firm**
→ **Hard**
→ **Absent**

Surface → **Smooth**
→ **Nodular**
→ **Irregular**

Mass: Present (or) Absent

Free Fluid: → **Present**
→ **Absent**

Guarding → **Present**
→ **Absent**

Rigidity → **Present**
→ **Present**

c. Percussion

Liver Dullness **Normal**

Over The Mass → **Dull**
→ **Resonant**

Free fluid → **Shifting Dullness**
→ **Puddle's Sign.**

d. Auscultation:

Bowel Sounds → **Present**
→ **Absent**

Systemic Examination

1. **CVS**
2. **Rs (1) Respiration** → **(Normal)**
→ **Restricted**

Inter costal → **Fullness**
→ **Tenderness**
→ **Edema**

percussion → **Resonant**
→ **Impaired**
→ **Dull**

Provisional Diagnosis

Differential Diagnosis:

Benign **(1)** **Acute Cholecystitis**
(2) **Viral hepatitis**

(3) **Hydatid Cyst**

(4) **Simple Cyst**

Malignant (1) **Hepatoma**

(2) **Secondary Liver**

Investigation

1) **Hb%**
2) **TC, DC, ESR**
3) **Urine** → **Albumin**, **Sugar**, **Deposit**
4) **Stool** → **Oval**, **Cyst**, **Trophozoites**
5) **Pus Culture & Sensitivity**
6) **Serology (IHA – ALA)**
7) **LFT**
8) **X-ray Chest PA view**
9) **X-ray Abdomen erect**
10) **USG**
11) **CT**

Analysis

Total Patients Studied – 106

ALA = 93 (87.7%)

PLA = 13 (12.3%)

Treatment

1) Drug Therapy

2) Needle aspiration

3) PCD

4) Open Surgical Drainage

5) Complication.

Annexure III

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